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# HANDBOOK

OF

# PRACTICAL MEDICINE

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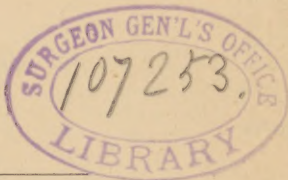
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VOLUME I

DISEASES OF THE CIRCULATORY AND RESPIRATORY APPARATUS



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ONE HUNDRED AND THREE WOOD ENGRAVINGS

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# HANDBOOK OF PRACTICAL MEDICINE.

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## SECTION I.

### DISEASES OF THE CIRCULATORY APPARATUS.

DISEASES of the circulatory apparatus are associated to a great extent with certain mechanical disturbances. This is owing to the fact that the organs in question mainly subserve mechanical purposes, inasmuch as they maintain the regular rhythm of the circulation by certain pump arrangements and canals.

We can readily understand, therefore, that these very diseases can be recognized with great certainty by the aid of physical methods of examination. Only those who are masters of such methods of examination will feel themselves at home in this class of diseases.

## PART I.

### DISEASES OF THE PERICARDIUM.

#### 1. *Inflammation of the Pericardium—Pericarditis.*

I. ETIOLOGY.—With regard to etiology we distinguish two forms of pericarditis, the primary (idiopathic, protopathic), and the secondary (deuteropathic, symptomatic). Primary pericarditis is restricted almost entirely to those cases in which the inflammation of the pericardium directly follows an injury which has affected the region of the heart (usually a fall, blow, push, or contusion).

A rheumatic pericarditis, caused by a cold, was formerly included in this category. In accordance with the well-founded attempt of modern medicine to restrict the etiological potency of rheumatism, and to explain much that was attributed formerly to cold, to the action of certain lower organisms—bacteria, schizomycetes—there is a just doubt

whether a contagium vivum does not play a part also in rheumatic pericarditis. However, this form of pericarditis does not occur very often. It is observed with relative frequency in the autumn and spring months, and it has been found that a number of such cases may occur within a short period, *i. e.*, almost like an epidemic.

Secondary pericarditis is by far the more frequent form. That variety which develops in the course of acute articular rheumatism occupies the first rank in point of frequency. Adults and men are affected so often with pericarditis for the reason that children and women suffer less frequently from acute articular rheumatism. Von Bamberger has called attention to the fact that pericarditis is associated with special frequency with those cases of articular rheumatism in which either many joints are affected, or the morbid changes rapidly pass from one joint to another.

The statements concerning the frequency of pericarditis in acute articular rheumatism vary from 14 per cent (v. Bamberger) to 37.3 per cent (Ormerod) of all the cases, but the experience is general that children suffering from acute articular rheumatism run a special risk of the development of pericarditis as a complication. It begins most frequently from the fourth to the fourteenth day of the disease, but there are rarer cases in which it precedes the articular affection. Since acute articular rheumatism is undoubtedly an infectious disease, and is caused by certain bacteria, we may assume the following connection between it and the pericardial inflammation, *viz.*, that the low organisms are carried through the general circulation into the pericardium and there also give rise to inflammation. In view of the great similarity in structure of the pericardium and the synovial membrane of the joints, and the close relationship of their physiological function, it is not astonishing that one and the same morbid agent should so often affect both organs.

According to Charcot, pericarditis is associated also with chronic articular rheumatism.

Secondary pericarditis is also observed in many other infectious diseases—in pyæmia, septicæmia, relapsing fever, typhus, more rarely in typhoid fever, also in cholera, variola, dysentery, scarlatina, morbilli, erysipelas, diphtheria, cerebro-spinal meningitis, malaria, and, according to some, in gonorrhœa and syphilis (pericarditis gummosa). Perhaps we will not go astray in these cases, also, if we assume a direct action of the schizomycetes which give rise to the diseases mentioned. Bednar asserts that he has observed pericarditis a number of times after vaccination.

In some cases the development of pericarditis is owing to certain constitutional diseases. It is observed often in acute and chronic Bright's disease. It occurs not infrequently in cancerous cachexia, or advanced pulmonary phthisis; also among drunkards, in scurvy, morbus maculosus Werlhofii, purpura hæmorrhagica.

Finally, there is another group of cases which is associated with inflammations of adjacent organs, the inflammation being propagated directly. Thus pericarditis not very rarely follows affections of the endocardium, or the heart muscle. This may also happen in aneurisms of the aorta and pulmonary artery. Pericarditis also develops not infrequently in the course of pleurisy and fibrinous pleuro-pneumonia, particularly after left-sided pleurisy. Furthermore, very superficial cavities in the lung may give rise to pericarditis, and this is inevitable

if, after adhesion of the surfaces, the contents of a cavity rupture into the pericardium. In the same way pericarditis may develop in caries of the ribs, sternum, or vertebra, in suppuration of the bronchial and mediastinal lymphatic glands, as the result of perforation of the pus into the pericardium, and the formation of an internal pericardial fistula. Diseases of the œsophagus occasionally produce pericarditis. This occurs particularly in cancerous processes which extend to the pericardium, and in ulcerations produced by ingested foreign bodies, which lead to perforation of the œsophagus and pericardium. Diseases of the abdominal organs may also give rise to pericarditis. This category includes peritonitic exudations, which have become encapsulated near the diaphragm, abscesses and echinococci of the liver and spleen, inflammations of the capsule of the liver, ulcers and cancers of the stomach, the former of which rupture into the pericardium with comparative frequency, and tumors of other abdominal organs. Finally, inflammations of the skin or diseases of the mammae may spread to the pericardium. Pericarditis develops most frequently between the fifteenth and thirtieth years of life. It is rarely observed in children, especially before the age of six, though cases of fetal pericarditis are reported. Men are affected more frequently than women, evidently because they are more exposed to the exciting causes.

**II. ANATOMICAL CHANGES.**—Pericarditis is divided into a circumscribed and diffuse form, according to the extent of the anatomical process. In the former variety, the changes are usually confined to the base of the heart and adjacent large vessels. In diffuse pericarditis the inflammation is almost always more marked upon the epicardium than upon the parietal layer. We will describe only the latter form, as it differs anatomically in no respect from the former.

The first appearances are unusual redness and injection. The vessels of the subserous connective tissue and the serous membrane itself are visible in many places as stellate or dendritic vessels, while in other places the hyperæmia appears as a diffuse, uniform redness. Small extravasations of blood are usually seen in a few spots.

The surface of the pericardium very soon loses its gloss. As a result of the swelling and partial desquamation of the endothelium, it appears roughened to a certain extent, as if breathed on, or like velvet. It can also be noticed that the tissue of the pericardium is loosened by exudation.

The first products of inflammation are soon deposited upon the surface of the pericardium. In the beginning they are thin cobweb-like, or gauze-like membranes, which are readily scraped off with the blade of the knife. They increase gradually in size, and are converted into yellow or grayish-yellow, rind-like masses of fibrin, several millimetres in thickness, and which, upon opening the pericardium, usually present a very uneven surface, either like a network or raised in numerous parallel rows. This surface has been compared sometimes to the porous appearance of a sponge, to a honeycomb, or to the appearance presented by the inside of the second stomach of a calf, or to the outside of a pine cone, or finally to two pieces of buttered bread which have been pressed against one another and then torn apart. The appearance described is evidently produced in the same way, inasmuch as the masses of fibrin situated on the epicardium and pericardium are rubbed against one another by the movements of the heart. Sometimes the surface of the heart appears to be covered with numerous villi (*cor villosum* s. *hirsu-*



tum, villous heart, hairy heart). This anatomical form of pericarditis is known as dry fibrinous pericarditis.

As a rule, in addition to the formation of fibrinous masses, there is also a fluid exudation. According to its character, we distinguish a serous, purulent, and bloody exudation, so that we may speak of pericarditis serosa, purulenta s. pyo-pericardium, and hæmorrhagica.

Pericarditis serosa (properly sero-fibrinosa) presents a slightly cloudy fluid, poor in cells, which has a grayish-yellow or grayish-green color, and is interspersed with small, more or less profuse masses of fibrin.

In pericarditis purulenta, the fluid is rich in cells, and presents the opaque, greenish-yellow constitution of pus. It may exceed three liters in amount. In such cases, after removal of the sternum and adjacent cartilages of the ribs, the intact pericardium forms an elongated, round, tense body, in which fluctuation is made visible upon slight shock. It is evident that considerable enlargement of the pericardium will have an effect upon adjacent organs. The lower lobe of the left lung in particular is often compressed into a small space, and deprived of air. But we rarely have to deal with a purely purulent, but usually with a fibrino-purulent pericarditis, the fibrin being deposited in part upon the surface of the pericardium, in part freely movable in the fluid in the form of flakes and threads. Purulent pericarditis develops especially when the inflammation is due to pyæmic processes, or to the perforation of pus from neighboring parts.

Pericarditis hæmorrhagica arises usually from dyserasic causes. It may be looked for in cancer and tuberculosis, scurvy and morbus maculosis Werlhofii, the hemorrhagic forms of variola, morbilli, scarlatina, more rarely in Bright's disease. In recent cases, the pericardium is filled with unchanged and partly coagulated blood, the quantity of which may be so great that the other internal organs are strikingly anæmic. In older cases, we find a brownish-red fluid, in which the red blood-globules are dissolved in great part, and their coloring matter set free. There are also various combinations and gradual transitions to other forms of pericarditis. Thus, in fibrinous pericarditis the fibrin is often speckled in a striking manner with dots of blood, and a serous or purulent exudation may also be colored more or less red from an admixture of blood.

Like all other inflammatory processes, those of pericarditis depend in the main upon the blood-vessels and their contents. Dilatation of the vessels, exudation of the fluid constituents of the blood, emigration of white blood-globules, rhexis of the vessels, and diapedesis of red blood-globules are the chief factors. According to the researches of Chapman, Muench, and Rindfleisch, a portion of the pus corpuscles in the exudation is produced by proliferation of the nuclei of the endothelium of the pericardium. Under the microscope, the fibrin is found in the shape of intertwined threads or granular masses which include round cells in greater or less number. The cells are especially abundant in the deeper layers of the fibrin which is situated on the surface of the pericardium. The tissue proper of the pericardium is also infiltrated with pus corpuscles, which are more abundant the more we approach the surface. Pus corpuscles form the chief cellular constituent of the serous and purulent exudations. Not infrequently they are in a condition of fatty degeneration. In addition, there are free nuclei, granular detritus, a few free drops of fat, or larger accumulations of fat. Here and there are elements of an endothelial character.

In a recent hemorrhagic exudation, the blood-globules are unchanged. After a time they are dissolved, and fatty cells and hæmatoidin crystals may form in the clots.

Peter reports that he observed thickening and extravasations in the neurilemma and granular degeneration of the medullary sheaths in the phrenic nerve, so far as it is connected with the pericardium.

Pericarditis presents manifold sequelæ and terminations. Changes can be detected almost invariably in the layers of the heart muscle next to the pericardium. They look peculiarly dull and pale, occasionally are speckled yellow and marbled, and are flabby and brittle. Under the microscope we recognize marked fatty degeneration, many fibres being so profusely infiltrated that their true structure is destroyed. Deeper down, the fatty degeneration passes gradually into a condition of cloudy swelling. The superficial layers of muscle present not infrequently a profuse infiltration of the connective tissue bands with round cells.

In purulent pericarditis superficial ulcerations may form upon the surface of the pericardium, and spread occasionally to the upper layers of the heart muscle. Perforation of the pus externally (total pericardial fistula) may also occur, though very rarely. The pus sometimes follows a very peculiar course. Sabatier observed perforation of the pus in the lower part of the neck, immediately beneath the left clavicle, and Fabricius described a perforation in the second right intercostal space. Rich recently reported a case in which perforation of the sternum occurred with the formation of a præsternal abscess as large as an apple, and Wyss had previously reported a case of pericardial fistula of two years' standing with perforation through the ribs.

Nor can we deny the possibility of the rupture of the pus into adjacent cavities and organs.

In certain cases the inflammation extends to the outer surface of the pericardium (pericarditis externa). The inflammatory process may thus extend to the mediastinal connective tissue, or the adjacent pleural surfaces. In the former event, the connective tissue may present merely a marked injection and moisture, but diffuse purulent infiltration or circumscribed abscesses may also be present.

A very dangerous condition develops, if pus, more rarely blood, decomposes in the pericardial cavity and becomes ichorous (pericarditis putrida). This is to be looked for, in particular, when air has entered the pericardium, when the pericarditis is caused by pyæmic or septic processes, or when decomposed and ichorous masses have ruptured into the pericardial cavity, as may readily happen in cancer of the œsophagus and stomach, or pulmonary cavities. Under such circumstances, the exudation has a nauseous, cadaverous odor. Under the microscope the cells are found, in great part, in a condition of granular degeneration. We also find a large number of schizomyxetes, frequently also delicate crystals of the fatty acids, grouped in places into sheaves or alveolar formations.

It is evident that the terminations and sequelæ of pericarditis cannot be separated sharply from one another. The most favorable termination, of course, is complete absorption of the exudation and a return to the normal, but this rarely happens.

Thickenings of the pericardium are left over in many cases. They constitute white, fibrous patches, which may be exceedingly hard and almost cartilaginous in consistence. They are termed tendon spots,

maculæ tendinæ, albidæ, lactææ, although the majority of them, as we will see later, are not purely inflammatory in their origin.

In other cases, the surface of the pericardium presents villous or polypoid connective-tissue formations, which occasionally are densely aggregated in astonishing numbers. It also seems as if constrictions sometimes occur in certain villi, and that the latter enter the pericardial cavity as free bodies.

Connective-tissue bands and membranes form very often in the course of pericarditis, and connect the opposed surfaces of the pericardium (pericarditis adhesiva). In such cases the fluid portion of the exudation is absorbed, the fibrin undergoes granular and mucoid degeneration, and disappears in part, while the cellular elements are organized into connective tissue, and are vascularized by the underlying pericardium and epicardium. These changes, if they are not too extensive, are capable of recovery, inasmuch as the synechiæ are stretched, thinned, and finally torn by the movements of the heart. We observe partial synechia most frequently in the vicinity of the large vessels.

The connective-tissue adhesions sometimes extend over the entire pericardium, giving rise to complete obliteration of the cavity (concretio pericardii).

If the absorption of the pericarditic exudation is not complete, and a free formation of fibrous adhesions takes place, the pericardial cavity is converted into a meshed space, the chambers of which are filled with fluid and partly solidified exudation.

If the exudation is purulent, the fluid constituents of the pus may be entirely absorbed, while the cellular remainder dries, disintegrates, and is converted into a cheesy mass. If this is infected with tubercle bacilli, numerous tubercle nodules sprout up and are especially abundant in the new-formed adhesions. In other cases the cheesy mass undergoes calcification, and if the latter is very marked, the entire heart may be inclosed in a sort of calcareous cloak.

Sero-fibrinous pericarditis is the most frequent form of the disease. Among 324 cases of pericarditis, upon which autopsies were made at the Berlin Charité from 1866-1876, Breitung found the following relations:

Pericarditis sero-fibrinosa, . . . . .	108
“ hæmorrhagica, . . . . .	30
“ purulenta, . . . . .	24
“ tuberculosa deuteropathica, . . . . .	24
“ tuberculosa idiopathica, . . . . .	2
“ adhæsiva partialis, . . . . .	111
“ totalis, . . . . .	23
“ ossificans, . . . . .	2
	<hr/>
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The other anatomical changes found in those who have died of pericarditis depend in part upon the primary disease, in part they are the result of venous stasis, as shown in congestion of the organs, œdema of the lungs, enlargement of the liver, etc.

III. SYMPTOMS.—Although it is usually very easy to recognize pericarditis by the aid of physical examination, it is impossible to do so from the subjective complaints and other clinical symptoms. Hence we should never fail to make a repeated examination of the heart in certain diseases, particularly in acute articular rheumatism.



The physical diagnosis of pericarditis is based on three cardinal symptoms: *a*, the presence of pericardial friction sounds; *b*, the shape of the area of cardiac dulness; *c*, the character of the apex beat.

*a*. We may diagnose pericarditis with certainty from the mere presence of a pericardial friction sound. For, although statements have been made that tubercle, cancer, milk-spots, and even calcification of the coronary arteries, and abnormal dryness of the surface of the pericardium have given rise to friction sounds, these conditions are so extremely rare that they do not change the diagnostic significance of pericardial friction sounds in pericarditis, and it may be maintained that almost all such sounds are pericarditic. The acoustic character of the sound varies. In many cases it is a brief, gentle rubbing, as if the finger is passed lightly over taffeta or silk paper; in others it has a strikingly hard, dry, creaking character, reminding us of the creaking of a snowball, or of a bent piece of new leather. If the sound is very loud, it may be heard at some distance from the thorax. Under such circumstances it is felt not infrequently as fremitus, or the patients state that they experience a sensation of rubbing in the cardiac region. If large, smooth-walled cavities are near the heart, for example, pulmonary cavities, pneumothorax, stomach and transverse colon distended with gas, the resonance may impart a metallic quality to the friction sound.

The pericardial friction sound is characterized in many cases by the fact that it is not continuous, but is composed of several parts. As Traube has shown, we can generally distinguish three interruptions during a complete heart beat, one of which is presystolic and coincident with the contraction of the auricles, while the two other louder ones are synchronous with systole and diastole of the ventricles. At times, some parts are interrupted still further and produce, to a certain extent, the impression of fine râles (evidently adhesion sounds). It must also be mentioned that the intensity of the sounds presents striking changes. Stokes drew attention to the fact that, upon strong pressure with the stethoscope, the friction sounds become louder, inasmuch as the surfaces of the pericardium are thereby approximated, and the friction is facilitated. As a matter of course, this phenomenon is more distinct the more flexible and yielding the thorax is (in children and females). Under certain circumstances friction sounds may be produced temporarily by pressure, although without it no abnormal sounds are heard, and the pericarditis has apparently run its course. The pressure should not be excessive, however, for Friedreich has shown that this interferes with the movements of the heart and diminishes the intensity of the friction sound. The intensity of the sound is also greatly influenced, as a general thing, by the position of the body; so that it may be heard only in the sitting position or when the body is bent over forwards, and may be absent in dorsal decubitus. This is owing to the fact that the layers of the pericardium are approximated in a different degree in the various positions of the body. Finally, the intensity of the friction sound is affected by the respiratory movements, being increased during inspiration and diminished during expiration. This is probably explained by the fact that, on account of the inspiratory distention of the lungs and contraction of the diaphragm, the surfaces of the pericardium approach one another, so that more vigorous friction is facilitated. Exceptions to this rule are rare. For example, Lewinski has described a case in which, on account of adhesions between the pulmonary and mediastinal pleura, the pericardial friction sound was increased during expiration.



We must be careful not to estimate the intensity and extent of the inflammatory process from the intensity of a pericardial friction sound. In persons who have died of Bright's disease, I have observed several times that very loud friction sounds corresponded to very slight and circumscribed changes in the pericardium. Even when the friction sound is very loud, it very rarely, as opposed to endocardial murmurs, extends beyond the region of cardiac dulness. Indeed, in not a few cases it is confined to a space hardly as large as a thaler, so that it is inaudible even a centimetre beyond this spot.

The development of the pericardial friction sound is readily understood. It is evidently owing to the fact that the surfaces of the pericardium are deprived of endothelium and have become roughened, so that during the movements of the heart, the rubbing of the rough surfaces produces a scratching, scraping sound. Friedreich has shown that roughness of one pericardial surface suffices to produce a friction sound. As the first inflammatory changes develop commonly at the base of the heart and near the great vessels, it is not astonishing that friction sounds are heard there first, or exclusively. They are most frequently heard, therefore, over the middle third of the sternum and immediately adjacent to the left edge of the sternum, in the third and fourth left intercostal spaces. As a matter of course, the friction sound can only be produced where the pericardial surfaces are in contact with one another. If they are separated by fluid exudation, the possibility of its development in such places is lost. But this does not mean that a friction sound is never heard when fluid exudation is present in the pericardium, since the conditions for its development may be present above the fluid. According to Cejka, a friction sound may be heard and felt when the pericardium contains as much as 1,000 ccm. of fluid. In exudative pericarditis a friction sound is produced most frequently at the beginning and end of the disease. In the latter event it is a favorable prognostic sign, since it indicates the occurrence of absorption. Nothing can be predicated concerning its duration; sometimes it lasts a few minutes or hours, sometimes many weeks.

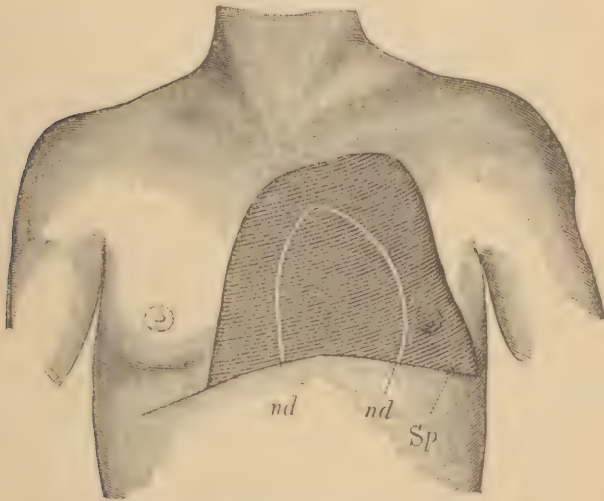
*b.* The area of cardiac dulness assumes a shape which is characteristic of pericarditis when the inflammation has led to the formation of a large amount of fluid exudation. This symptom, therefore, is opposed, to a certain extent, to that just described; for while the dulness changes the more the greater the amount of fluid in the pericardium, the latter condition is apt to cause disappearance of a friction sound.

The shape of the area of cardiac dulness depends on two circumstances. The greater the amount of fluid in the pericardium, the more its size increases, and, with it, the extent of the cardiac dulness. But we must not forget that in a pericardium filled with fluid, the heart, as the heaviest part, occupies the lowest position in all positions of the body. The minimum amount of fluid necessary to produce a change in the cardiac dulness varies partly according to the individual, and also according to the condition of the adjacent organs, *i. e.*, according to the mobility of the anterior borders of the lungs. Under especially favorable circumstances it is possible to determine by percussion the presence of 100 ccm. of fluid.

If fluid exudation is formed, it accumulates first at the base of the heart between the anterior parietal layer of the pericardium, and the large vessels. Almost at the same time there is an accumulation of fluid along the diaphragmatic part of the pericardium. This change is recog-

nized on percussion by the fact that the greater (relative) cardiac dulness extends at its lower border, beyond the left mammary line and the right edge of the sternum, and that a very marked and extensive dulness also appears at the base of the heart. The more the fluid increases in amount within the pericardium, the more it spreads in all directions, extending between the anterior surface of the heart and the pericardium, and thus separating the heart from the anterior thoracic wall. The anterior borders of the lungs are also dislocated, being pushed to the outside by the distended pericardium. A greater part of the anterior pericardial surface than under normal conditions is therefore in contact with the inner surface of the thorax. The cardiac dulness therefore extends in all directions. Superiorly, it may extend to the second, even the first left costal cartilage; on the right side it may pass beyond the mammary line, and on the left side it may touch the axillary line. It also enlarges

FIG. 1.



Cardiac dulness in pericarditis of more than moderate amount. *Sp*, Apex beat. The white line *nd* indicates the normal boundaries of the large (relative) area of cardiac dulness.

inferiorly, so that the lower border of cardiac dulness may extend to the eighth left rib. Under such circumstances the pulsating heart may be occasionally reached with the fingers from the left hypochondrium and the epigastrium, and is felt as a rhythmically pulsating, projecting tumor. The semilunar space must therefore diminish in size. The shape of the cardiac dulness changes from the normal triangular to a quadrilateral or, properly speaking, trapezoidal figure (vide Fig. 1). The right and left sides of the figure pursue a different course, the former passing abruptly from above downwards, the latter inclining more gradually. It must also be noted that, according to Gerhard, the extent of the cardiac dulness varies in the erect and recumbent positions, inasmuch as it may increase one-third to one-half in height in the former position.

The small (absolute) cardiac dulness and the cardiac resistance also increase in exudative pericarditis.

Extensive exudation into the pericardial cavity may be detected by other methods of physical examination than percussion.

On inspection, the præcordial region is often found enlarged, indeed the enlargement (voussure) extends not infrequently beyond the region of the heart, embracing a large part of the left thorax and a part of the right half of the thorax lying next to the sternum. The dilated portions are noticeable on account of the dilatation of the intercostal spaces; the left nipple is higher than the right; the skin is peculiarly tense, devoid of folds and shining, and, if the fatty layer is not too thick, the subcutaneous veins appear like bluish strands. The dilated portions take very little or no part in the respiratory movements. As a matter of course, the dilatation is so much more marked the greater the amount of exudation and the more yielding the thorax, so that it is especially well marked in children and women. But it must not be forgotten that the dilatation is not an exclusive effect of pressure. We must also take into consideration the inflammatory paresis of the intercostal muscles, since the dilatation is sometimes greater than we would expect from the amount of fluid.

In many cases the movement of the heart is not visible, in others a diffuse systolic elevation of the entire præcordial region is noticeable. It sometimes happens that a sort of wave movement is observed if the intercostal muscles are broad and thin, and this cannot be attributed to a directly visible movement of the heart muscle, but is dependent on a wave-like movement of the fluid. This phenomenon is so rare that very experienced writers have denied—though erroneously—its existence.

The position of the patients must also be noticed. In almost every case they assume an elevated dorsal position, usually inclined to the left side, while they suffer from dangerous dyspnoea in the recumbent posture, or when lying on the right side. This is not astonishing in view of the fact that in the right lateral position the right lung is hindered in free respiration on account of the weight of the body, while the left lung in all cases takes very little or no part in respiration. Very unusual positions of the body are sometimes assumed. In one case the patient could combat the dyspnoea only by bending forward on his hands and knees.

On palpation we are struck with the fact that the integument of the præcordial region can be raised into folds less readily than on corresponding parts of the right half of the thorax. In a few cases slight depressions in the skin remain after pressure, showing the existence of slight oedema. But we may not conclude therefore, that the pericardial exudation is purulent, for it is often merely an inflammatory collateral oedema caused by the pericardial inflammation. Vocal fremitus is absent in the region of dulness, or is felt very feebly at its borders. Pressure upon the dilated portion of the thorax does not always give rise to pain. Some authors state that they have felt fluctuation in the intercostal spaces.

Peter has attached importance recently to a local elevation of temperature in the præcordial region. While the temperature of the skin in this locality in healthy individuals is  $35.8$ – $36^{\circ}$  C., in pericarditis it may reach  $37.8$ – $38.7^{\circ}$  C. and occasionally exceeds the axillary temperature.

Among the phenomena recognized on percussion, we must also consider those produced by pressure on adjacent organs. The lower lobe of the left lung is not infrequently compressed to such an extent that a dull, or dull tympanitic percussion note is heard over the lower half of



the left dorsal and lateral surfaces. The compression may be so great that the left upper lobe projects like a hernia above the clavicle. Pericarditis is readily differentiated from left-sided pleurisy with effusion by the fact that the vocal fremitus is not diminished, as in pleurisy, but increased. Mistaking it for pneumonia can also be avoided, for, although dulness and bronchial breathing are common to both affections, consonant râles are absent in simple compression of the lungs. It should also be mentioned that at the periphery of the cardiac dulness the transition from the flat percussion note to the loud pulmonary note is effected by means of a tympanitic note, which is also explained by compression of the anterior border of the lungs. A slight, dull tympanitic percussion note is heard occasionally at a very early period in the first and second left intercostal spaces, and this may precede for several days the development of the characteristic cardiac dulness. Percussion shows not infrequently that the left lobe of the liver is pushed downwards, the result, evidently, of the unusual weight of the pericardium.

Upon auscultation, we are struck most forcibly by the fact that the heart sounds are extremely weak because the fluid on the anterior cardiac surface impedes the conduction of the sounds to the thoracic walls. Gendrin states that agophony is heard on auscultation of the præcordial region when the patient is sitting erect and slightly bent forward. But in such cases the amount of fluid must be considerable.

*c.* The apex beat will be characteristic of pericarditis only when the pericardium contains fluid exudation. This is shown in two ways, by the gradual disappearance of the apex beat, and also by the fact that cardiac dulness extends to the left, beyond the apex beat.

The gradual disappearance of the apex beat is owing to the fact that the fluid, the more it accumulates, inserts itself between the anterior surface of the heart and the wall of the thorax. The apex beat may disappear in the absence of pericarditis, when the contractions of the heart lose their power, but the pulse then becomes very small and feeble, in contradistinction to what occurs in pericarditis. In exudative pericarditis the apex beat can be felt not infrequently when the patient sits erect and slightly bent forward, the heart being thus brought in contact with the thorax.

The cardiac dulness extends beyond the apex beat because the distended pericardium no longer confines itself to the boundaries of the heart, but extends beyond them in all directions.

A less characteristic peculiarity of the apex beat is its excessive capacity for dislocation in a lateral position of the body. This is the result of the unusually large dimensions of the pericardium, and of the fact that the heart, on account of its weight, falls, in a lateral position of the body, in a corresponding direction.

The remaining symptoms of pericarditis are by no means so important as those just described. The least reliable are the subjective symptoms, and physicians connected with large hospitals will have found at times that patients with large pericardial exudations enter the hospital, complaining indeed of feeling sick for some weeks, but still able to go about, and compelled only recently to enter the institution on account of increasing dyspnoea.

Pain in the præcordia develops not infrequently during pericarditis. It is most severe when the fluid exudation is small in amount, while it often moderates into a feeling of dull pressure and tension when the accumulation of fluid is considerable. The pain occasionally radiates in



different directions so that the patient is annoyed by painful sensation in the back, left arm, even in the left auricular region, epigastrium, and umbilical region.

The patients complain usually of palpitation of the heart, which is either constant or appears after slight motion, often after a change of position. It is usually associated with a feeling of oppression, anxiety, and dyspnœa.

The dyspnœa may also be constant. Pericarditis impedes the action of the heart in many ways. We have already referred to the fact that the heart muscle does not remain unaffected by the inflammation of the pericardium. In addition, a copious exudation exercises pressure on the heart. This affects particularly the thin-walled venæ cavae and auricles, giving rise to venous stasis. But the large arteries are not unaffected, and the arterial efflux from the heart is obstructed. The dyspnœa is also increased by the compression of the adjacent portions of the lungs.

Profound syncope occurs occasionally, especially when the patients suddenly assume an upright position (anæmia of the brain), and this may be the immediate cause of death. In other cases, signs of cerebral congestion appear; the patients grow drowsy and stupid, become delirious, and are attacked occasionally with convulsions—symptoms which present a grave prognosis and not infrequently precede death for a short time.

In rare cases hoarseness is produced, and is shown by the laryngoscope to be the result of paralysis of both vocal cords. In other cases the left vocal cord alone is paralyzed, a phenomenon readily explained by pressure of the exudation on the left recurrent nerve. If the right recurrent is also paralyzed, we may suspect compression by the enlarged veins.

Disturbances of deglutition have been observed much more frequently. They could rarely be attributed to compression and stenosis of the œsophagus by the distended pericardium. In certain cases they seemed to be the result in part of inflammation of the pneumogastric, in part of inflammation of the muscular coat of the œsophagus. Guérin mentions that certain patients were affected with pharyngeal spasm at the mere sight of fluids (pericarditis hydrophobica).

Bourceret proved by experiments on animals that, in addition to the pneumogastric, the phrenic nerve may also be inflamed in pericarditis. To this we must attribute, in the main, the hiccough and vomiting, though the mechanical irritation of the diaphragm and stomach by the abnormally heavy pericardium must not be underestimated.

When the sensation of pain is absent, it may be produced not infrequently by pressure in the region of the heart. Greater importance was attached to the pain produced by pressure in the epigastrium between the xiphoid process and the left hypochondrium, more rarely on the right side. Guéneau de Mussy states that it may precede all other symptoms of pericarditis, and is more constant than the pain in the præcordial region. He attributes this pain and also the sensitiveness at the lower part of the flexors of the neck to inflammation of the phrenic nerve.

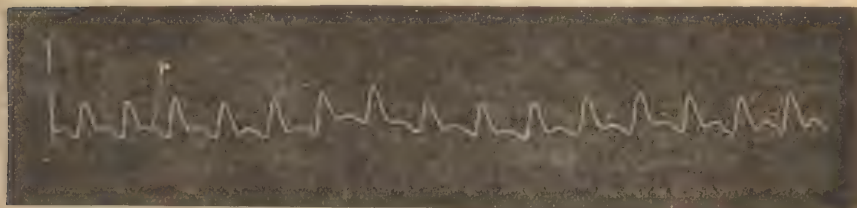
Fever may be entirely absent, even in purulent pericarditis, but in the majority of cases the bodily temperature is elevated, because the primary disease is usually associated with fever. In the latter event, the pericarditis may only be made manifest by the fact that the fever rises to an unusual height.

The pulse is usually very rapid at the onset, and is often irregular.

without losing strength. This phenomenon is properly attributed to inflammatory irritation of the ganglionic nervous apparatus within the heart muscle. The irregularities of the pulse occasionally assume that form known as *pulsus bigeminus*, and *pulsus alternans*. Retardation of the pulse may be looked for when the trunk of the pneumogastric is implicated in the inflammatory process directly or by pressure. After the disease has lasted for some time the increased rapidity of the pulse usually continues, but it generally loses considerably in strength, as the result of diminished energy of the heart. In some cases the radial pulse has been found to become small or disappear during deep inspiration (*pulsus paradoxus* s. *pulsus inspiratione intermittens*). Traube also noticed in one patient that the left carotid and radial arteries were considerably narrower than the corresponding vessels on the right side.

With the aid of Marey's sphygmograph I have often followed the changes in the pulse for weeks. The recoil elevations are usually very marked, so that a typical dirotic pulse may result, while the elevations of elasticity become less or disappear. The pulse curve indicates, therefore, considerable diminution of vascular tension.

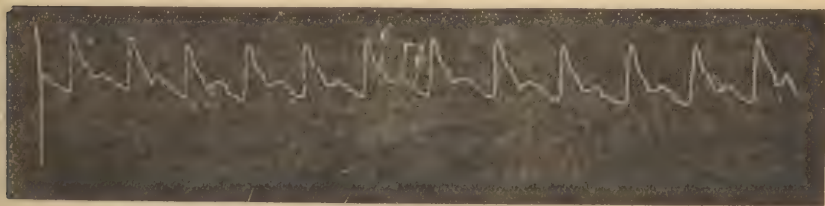
FIG. 2.



Pulse curve of right radial artery in sero-fibrinous, apyrexial pericarditis. Third day of the disease. Axillary temperature  $37.3^{\circ}\text{C}$ .

Fig. 2 is taken from a case in which the pericarditis followed a slight left-sided pleurisy. The disease was apyrexial, lasted seventeen days, and the sphygmographic examinations were made daily. The elevations due to elasticity gradually made their appearance a long time after the disappearance of the pericarditic symptoms. (Compare Fig. 3.)

FIG. 3.



Pulse curve of right radial artery in same patient. *r*, elevation of recoil; *e'e''*, first and second elevations of elasticity.

As a rule, the cervical veins are extremely dilated, a proof of the obstruction of the venous flow to the heart. They often present undulatory movements, and Stokes and Friedreich claim to have observed a

true venous pulse. But Riegel has recently emphasized that this cannot be a recurrent or positive wave of blood in the cervical veins, but is a negative venous pulse, produced by the temporary stasis in the outflow from the veins associated with the movements of the heart.

Systolic cardiac murmurs are heard not infrequently. They may be the result of the fever, degeneration of the heart muscle, compression of the large arteries, or a complication with endocarditis. Skoda mentions that he has sometimes heard a reduplication of the second sound over the aorta and pulmonary artery, and, according to Cejka, the first aortic sound is occasionally absent—a phenomenon attributed by him to inflammatory changes in the walls of the aorta.

Stasis in the veins is also shown by the color of the skin, the cheeks, lips, and visible mucous membranes presenting a cyanotic discoloration. At the same time the skin is usually pale, and if a large hemorrhagic exudation has occurred suddenly (as in scorbutus), the patients are as pale as a corpse, and look like one dying from hemorrhage.

The respirations are almost always accelerated. They are usually superficial, and accompanied not infrequently by expiratory moaning. Attacks develop occasionally in which the dyspnoea reaches an unusual height, so that the majority of the auxiliary muscles of respiration are brought into play. The alae nasi are dilated shortly before each inspiration, the larynx descends, and the contracted flexors of the head appear as firm, rigid strands under the skin of the neck. The expression of the face is anxious, and distinctly manifests great terror. The urine is generally scanty, has a saturated color, strongly acid reaction, and high specific gravity. Upon cooling, it very often deposits a reddish, granular, brick-dust-colored sediment, which consists of uric acid and urates, and is readily dissolved on the application of heat. Traces of albumin are found not infrequently. The albuminuria increases when the heart muscle becomes incapable of function, and casts are then found in the urine.

Symptoms of stasis are also manifested by the development of oedema, enlargement of the liver, ascites, hydrothorax, bronchial catarrh, occasionally hemorrhagic infarctions (hamoptysis). Finally, the patients, if our measures are unsuccessful, die with symptoms of pulmonary oedema, or the previously mentioned symptoms of cerebral congestion, or the increasing external and internal dropsy.

With regard to the extent of the inflammation, we distinguish circumscribed and diffuse pericarditis, clinically as well as anatomically. But we must not infer, in those cases in which the disease is manifested only by a very circumscribed friction murmur, that the inflammation is restricted to this locality, for the autopsy usually shows that the inflammatory process extends far beyond it.

With regard to the duration of the disease, we distinguish acute and chronic pericarditis, though there is no sharp boundary between these forms. Under certain circumstances, pericarditis is rapidly fatal. In the scorbutic form, which occurs particularly in the northern provinces of Russia, death has been observed within twenty-four hours. Pericarditis with a slight effusion of fluid may recover in a few days. The average duration of acute exudative pericarditis is one to two weeks. If it lasts more than four weeks it may be termed chronic, though partial absorption of the exudation not infrequently occurs temporarily.

The recovery of pericarditis is shown by the gradual diminution of the febrile movement, the amelioration of the respiratory disturbances,



the resolution of the local changes in the heart, and profuse diuresis. Some authors maintain that distinct retraction of the præcordial region is visible after absorption is completed. A striking irritability of the cardiac nervous system persists for a long time, so that slight bodily or mental exertion accelerates the movements of the heart to an excessive degree, and renders them irregular.

IV. DIAGNOSIS.—The recognition of pericarditis is based on the three cardinal symptoms, and is not difficult in the majority of cases. Errors result when one of the three symptoms is mistaken for some similar one, or when, for some reason, their development is impossible.

*a.* Pericardial friction murmurs may be mistaken for endocardial murmurs. A mistake is especially apt to be made when the friction murmur is gentle and soft, or the endocardial murmur is very harsh. In many cases, however, the character of the murmur is decisive, the pericardial murmurs being dry and rubbing. Furthermore, the latter appear to be superficial, while endocardial murmurs, to a certain extent, come from the deeper parts. Pericardial murmurs are intensified by moderate pressure, while endocardial murmurs are unaffected; upon excessive pressure, both kinds of murmurs are enfeebled, on account of interference with the movement of the heart. During deep inspiration, endocardial murmurs almost always diminish in intensity, while pericardial murmurs are intensified. But exceptions to this rule occur, and Traube showed that endocardial murmurs may be increased in inspiration on account of an increase of the intracardial blood pressure, while Lewinski, on the other hand, observed expiratory increase of pericardial murmurs when extra-pericardial adhesions of the lungs were present. The occurrence of murmurs only in a certain position of the body, particularly when erect and bent slightly forwards, favors the diagnosis of pericardial murmurs, though this phenomenon is also observed, in rare cases, in endocardial murmurs. But special attention should be paid to the rhythm of the murmurs. While endocardial murmurs are associated strictly with the different phases of the cardiac revolution, and are strictly diastolic, systolic, or presystolic, pericardial murmurs drag along after the different phases, and not infrequently are inserted between them. It may also be mentioned here that it is not rarely impossible to hear endocardial murmurs alongside of pericardial murmurs, and that the former often are not audible until the latter have disappeared.

Mistakes in the localization of the friction murmur must also be avoided. If the pleural or peritoneal surfaces in the neighborhood of the pericardium are roughened, the movements of the heart may be communicated to them, and give rise to friction murmurs, associated with the cardiac movements. Murmurs of the former class are known as pleuro-pericardial or extra-pericardial murmurs. They are heard most frequently along the anterior border of the left lung, especially in the vicinity of the apex beat, *i. e.*, along the tongue-shaped prolongation of the left lung. It is characteristic of these murmurs that they are composed, to a certain extent, of two parts, one of which is associated with the movements of the heart, the other with the movements of respiration. During deep respirations they increase in intensity, and present the character of a pleural friction sound. If the respiration is checked at the height of deep inspiration, the murmur usually disappears in a very short time. If respiration is checked at the end of expiration, the pericardial element becomes very distinct, and during the next few contractions of the heart the murmurs seem to be entirely dependent upon them.



They disappear again after three to six cardiac contractions, and do not reappear until after renewed respiration.

A pericardiaco-diaphragmatic murmur in tubercular peritonitis has been described by Emminghaus, the friction sound being produced between the roughened surfaces of the diaphragm and liver by the contractions of the heart. The differential diagnosis depends on the absence of other symptoms referable to pericarditis.

Despite other favorable conditions, pericardial friction sounds will remain absent if extensive adhesions of the pericardium, particularly on the anterior surface, are present, as the result of previous inflammation. If we have reason to suspect the existence of pericarditis, we should not fail to search for friction sounds in different positions of the body, and under the application of pressure.

*b.* Diagnostic errors concerning the characteristic cardiac dulness may result from the fact that it is absent despite the presence of a large amount of fluid, or that it may be simulated by affections of adjacent organs.

The characteristic cardiac dulness remains absent if the anterior surface of the heart is adherent to the parietal layer of the pericardium. In such cases the exudation may collect in the posterior part of the pericardial cavity, which is converted occasionally into remarkably large cyst-like spaces. Difficulties in diagnosis are owing occasionally to the fact that the anterior edges of the lungs are adherent, so that they are not pushed back when fluid accumulates in the pericardium. Although, under such circumstances, the small (absolute) cardiac dulness does not increase, the large (relative) cardiac dulness, and the cardiac resistance, are unusually great, and a mistake is usually avoided by those who do not rely exclusively on the determination of the small cardiac dulness in the diagnosis of heart diseases. These remarks also hold good concerning the existence of advanced pulmonary emphysema.

Among diseases of adjacent organs, encapsulated pleurisy and infiltration of the edges of the lungs may simulate pericarditic dulness. But such dulness is usually more irregular and, in diseases of the pulmonary parenchyma, increased fremitus, bronchial breathing, and consonant râles readily prevent a mistake. Aneurisms and mediastinal tumors may also be a source of error, although the differentiation is not difficult if we consider the development of the disease and the other symptoms. Pericarditic dulness is distinguished from a considerable increase in size of the heart itself by the fact that the apex beat in pericarditis is usually feeble, or cannot be felt, and that it often does not reach the left outer boundary of cardiac dulness. In pericarditis, also, the cardiac dulness increases in height in the erect position, while it almost always remains unchanged when the heart increases in size. Furthermore, the intensity of the heart sounds is strikingly feeble in pericarditis. If we have observed the development of the disease, we will be struck with the shortness of the time in which the dulness changes, as opposed to muscular changes in the heart.

*c.* Disappearance of the apex beat in pericarditis is distinguished from conditions of heart failure by the character of the pulse, which retains its strength in pericarditis, and diminishes in strength in other cases.

The value of the fluid in the pericardium cannot be determined by physical examination. This is decided by clinical experience, though

mistakes cannot always be avoided. The primary affection must be taken especially into consideration. Pericarditis during acute articular rheumatism is usually sero-fibrinous. In pyæmic and septicæmic processes we must expect purulent, occasionally ichorous pericarditis. A hemorrhagic exudation can be looked for generally in scurvy, the hemorrhagic diathesis, cancer, and tuberculosis. But in many cases the question of the character of the fluid must be left open.

Finally, we must decide whether the fluid in the pericardium is the result of pericarditis or hydropericardium. The latter is negated by the presence of inflammatory symptoms (local and general) and the absence of causes of stasis and of other symptoms of stasis.

V. PROGNOSIS.—The prognosis depends on two circumstances, the nature of the primary disease and the extent of the inflammatory process. That form which develops during acute articular rheumatism has a relatively favorable prognosis, while it is very unfavorable when the pericarditis is the result of cancer or tuberculosis. The prognosis, furthermore, is usually unfavorable in pyæmic and septicæmic processes, **scurvy**, and **hemorrhagic exanthemata**.

Pericarditis as such may prove fatal in a very short time. This has been observed particularly in scorbutic pericarditis, in which the loss of blood should not be underestimated. In other cases, also, the extensive exudation may inhibit the circulation of the blood, so that death follows from excessive stasis in the venous system. Chronic pericarditis offers a more unfavorable prognosis than the acute form, because the danger of degeneration and loss of function of the heart muscle is very great, and the chances of absorption of the exudation become less with the longer duration of the disease.

Age, sex, and constitution are not without influence on the prognosis. The disease is most fatal in the first year of life (Gendrin) and in old age. It also appears to run a more unfavorable course in females than in males. It will not surprise us that it more often terminates fatally in feeble individuals than in strong, full-blooded ones.

VI. TREATMENT.—If there are no special complications, the treatment may be restricted to suitable dietetic arrangements and local antiphlogosis. The patients should be placed in a roomy chamber, which is aired several times a day by means of the adjacent rooms. Narrow, crowded apartments, the air of which is overloaded with carbonic acid, increase the symptoms. In winter, the temperature of the sick-room should be regulated by the thermometer and kept at 15° R. Excessive dryness of the air should be prevented by placing vessels filled with water upon the stove. We should give strict orders that the patient be prevented from sitting up, because fatal syncope may then result from cerebral anæmia. The patient should retain the recumbent position, and use the bed-pan in micturition and defecation. Straining in defecation should be avoided, and attention paid to securing regular evacuations. Enemata may be necessary for this purpose. The head should be kept as low as is compatible with the patient's symptoms, since this prevents most surely the development of cerebral anæmia.

During the first two weeks, the diet should be fluid (milk, eggs, meat-soups, glutinous soups of oatmeal or barley). Acid drinks, for example, lemonade, may be given to relieve thirst. Coffee and tea should be prohibited, as they often give rise to palpitation of the heart, but the cautious administration of beer and wine is allowable, if the patients have been addicted to them, and they do not give rise to any bad symptoms.

Cooked fruit, particularly apple-jelly, should be given at dinner, particularly if there is a tendency to constipation. If the disease is protracted for several weeks, the cautious use of beer and wine should be tried under all circumstances. It may then become necessary to give meat, preference being given to fowls (breast meat), venison, raw scraped beef, finely-scraped ham, veal cutlets prepared over a hot fire, sweet bread, and roast calves' brain.

For local antiphlogosis we may employ an ice-bag which, being placed on a piece of linen, should cover the region of the heart as much as possible. Excessive filling of the bag, very large pieces of ice, and great weight of the bag should be avoided. It should be refilled with ice at suitable intervals, otherwise the water will be quickly warmed after the ice has melted, so that the cold application is converted into a warm one, and increases the patient's sufferings. In the absence of ice, we may employ cold compresses, which should be carefully and rapidly changed. In addition to its purely antiphlogistic action, the application of cold moderates the action of the heart, so that existing palpitation is diminished or disappears and the action of the heart is slowed.

The therapeutic measures mentioned prove sufficient in not a few cases. If there is considerable acceleration of the pulse without diminution in its power, it is well to slow the action of the heart and afford rest to the heart muscle which has been involved in the morbid process, by the administration of large doses of digitalis.

We may prescribe the following:

℞ Inf. Digitalis.....	2.0 : 180
Kali nitric.....	10.0
Syrup. simpl.....	20.0

M. D. S. Take one tablespoonful every two hours.

If there is a tendency to constipation, kali nitricum may be replaced by an equal dose of tartarus depuratus, the mixture being well shaken and given in the same doses as the preceding prescription. The digitalis should be discontinued at once, as soon as its action is shown by the slowness and irregularity of the pulse, and the rule holds good here more strictly than in other cases, that the patients should be seen at least twice a day during the administration of the drug.

Veratrin and tartar emetic will also slow the pulse, but they should be avoided, because they give rise to collapse.

If the pains in the cardiac region are violent, it is advisable (in addition to the application of the ice-bag) to use leeches, from six to fifteen being applied to the region of the heart, according to the strength of the patient. Amelioration is produced not infrequently by dry or wet cups (five to ten) or the application of a large fly-blister to the cardiac region.

If violent and obstinate vomiting, hiccough, and disturbances of deglutition set in, or there is a marked feeling of oppression and insomnia, the administration of a narcotic is indicated. Subcutaneous injections of morphine may be used to advantage:

℞ Morphin. hydrochlorici.....	1.0
Aq. destillat.,	
Glycerini puri.....	āā 15.0

M. D. S. 2-5 minims to be injected subcutaneously.

As increased temperature may accelerate the pulse excessively and



favours degeneration of the heart muscle, high fever may be combated by the use of antipyrine (2.0 every hour until defervescence occurs, or, better still, 3.0–5.0 in an ounce of lukewarm water, given as an enema). Quinine (1.0–2.0) is less certain, while salicylic acid, salicylate of soda, and kairine not infrequently cause profuse sweats and symptoms of collapse.

If heart failure occurs temporarily, stimulants must be employed, viz., strong wine (port, sherry, Malaga, Madeira, Marsalla, 1–2 table-spoonfuls every quarter hour), brandy, Champagne, ether (5–10 drops on sugar every quarter hour), or camphor internally (0.05 in powder every hour), or subcutaneously:

R Camphoræ tritæ.....	1.0
Olei Amygdalar.....	10.0
M. D. S. $\frac{1}{2}$ –1 hypodermic syringeful subcutaneously.	

In persistent conditions of heart failure, we should prescribe nutritious food, beer or wine, and give digitalis in small doses, for example, fol. digitalis pulv., 2.0; kali nitric., 5.0; pulv. et succ. liquirit. q. s. ut f. pil. No. 30. D. S., 1 pill to be taken t. i. d. The drug should be discontinued as soon as its effect is shown by slowness and irregularity of the pulse.

When the symptoms of pericarditis have disappeared, the patient should not be allowed to get up too soon, as dangerous fainting spells sometimes occur during convalescence. Physical and mental excitement should be avoided for a long time, as they are apt to be followed by palpitation of the heart.

If the absorption of the exudation is tardy, absorbents should be employed locally and internally. Locally we may use tincture of iodine (tinct. iodi, tinct. gallarum, ãã 10.0), inunctions of the cardiac region with iodide of potassium ointment or iodoform ointment (iodoform, 5.0; vaselini, 5.0). Warm compresses and repeated blisters (v. Bamberger) are also used. Internally we may give small doses of iodide of potassium, for example, potas. iodid., 3.0; fol. digitalis pulv., 0.5; pulv. et succ. liq., q. s. ut f. pil. No. 30. D. S. (1–2 pills to be taken t. i. d.). If these remedies prove useless, we may resort to diuretics, laxatives, or diaphoretics. Diuretics are preferable, while diaphoretics, particularly pilocarpine (0.1 : 10; one syringeful subcutaneously) often produce dyspnoea, palpitation of the heart, and symptoms of collapse. We may specially recommend the subcutaneous administration of caffeine citrico-benzoicum (caffeine, aq. destil., glycerin., ãã 5.0, one syringeful subcutaneously morning and evening), and also adonis vernalis (inf. adon. vern., 5 : 150, one tablespoonful every two hours).

In cases in which death threatens on account of the excessive amount of exudation or in which absorption does not occur for a long time, paracentesis of the pericardium should be performed and the exudation removed artificially. If the results are not so fortunate as after the similar operation in pleurisy, this is evidently owing in part to the fact that the operation is often not performed until severe nutritive disturbances of the heart muscle have developed. In such cases the operation is attended with no special benefit, as it acts merely mechanically. It must not, therefore, be delayed too long. Roberts found 23 recoveries among 49 collected cases (47 per cent) and West found 36 recoveries among 79 cases (46 per cent). Surgical treatises should be consulted concerning the details of the operation, and particularly as to whether puncture, incision, or subsequent in-



jection of irritating fluids should be performed. We will merely remark that puncture should be performed when the exudation is serous, and best in the fifth left intercostal space, about 2 cm. from the left edge of the sternum; in purulent pericarditis incision is indicated.

The disease underlying the pericarditis occasionally requires special treatment. In pericarditis scorbutica we should give styptics (ergotin, acetate of lead, etc.) in order to check extravasation into the pericardial cavity.

## APPENDIX.

### *a. Pericardial adhesions. Synechiæ pericardii.*

1. Fibrous adhesions between the two layers of the pericardium very often persist after pericarditis. Their etiology is the same as that of pericarditis. But they are found not infrequently upon autopsy in individuals who never presented signs of pericarditis during life, and we may therefore infer that extensive pericarditis occasionally runs a latent course. In addition, we often find chronic affections of the respiratory organs, particularly emphysema, phthisis, pleural adhesions, or chronic Bright's disease.

Sero-fibrinous pericarditis seems most adapted to lead to pericardial adhesions. They are especially apt to develop if the inflammation is subacute from the start, and the action of the heart has been very feeble for a long time during the disease. They may develop very rapidly, as cases have been published in which the first signs of adhesion were noticed eight or nine days after the beginning of the pericarditic symptoms. They are observed not infrequently in the dead-house. Among 1,003 autopsies Lendet found them 61 times (6 per cent). According to Breitung's statistics, adhesions occurred 134 times among 324 cases of pericarditis (41.3 per cent).

2. According to the extent of the adhesions, we distinguish partial and total synechia.

Partial synechia occurs most frequently near the great vessels, sometimes as thin threads and strands destitute of vessels, sometimes as extensive, tense membranes.

In total synechia there may be entire disappearance of the pericardial cavity (obliteration or obsolescence of the pericardium). Sometimes the connective tissue between the epicardium and pericardium is loose and can be separated with the fingers, sometimes it is so rigid that it constitutes an indissoluble connection between the two layers. If the latter are not much changed, the condition may be interpreted falsely as absence of the pericardium. The layers of the pericardium often present considerable thickenings, so that they assume a rind-like and cartilaginous character. Between the adhesions are sometimes found spaces which are filled with thick cheesy or partly calcified masses—the remains of a preceding pericarditic exudation. In some cases the heart has been found inclosed almost hermetically in a calcified or ossified capsule, so that the possibility of movement of the heart is astonishing.

Adhesions outside of the pericardium are observed not infrequently at the same time. Anteriorly the pericardium may be adherent by firm connective tissue to the chest-wall; posteriorly there may be firm adhesions to the aorta, œsophagus, and spinal column; firm adhesions to the diaphragm have also been described. Adhesions of the anterior edges

of the lungs and the complementary pleural sinus, most frequently on the left side, have been observed.

The heart muscle is almost always changed. In it we often find fibrous bands (particularly in the layers immediately beneath the pericardium), brown atrophy and fatty degeneration of the muscular fibres, dilatation and hypertrophy (often confined to the right heart). These changes depend upon pericardial adhesions only when the latter compress the coronary arteries and thus interfere with the nutrition of the heart-muscle. In other cases the changes depend on previous pericarditis, valvular lesions, chronic diseases of the respiratory organs or kidneys. Hypertrophy in particular cannot be well explained solely by pericardial adhesions.

3. In very many cases pericardial adhesions are entirely unsuspected during life, and symptoms are often absent even in total obliteration of the pericardium.

The condition is only recognized under two circumstances, either when the power of the heart-muscle is diminished, or when the systolic locomotion of the heart downwards is diminished by the adhesions. In the former event functional changes, in the latter event physical changes will be produced. Functional disturbances may be looked for particularly when the adhesions are very firm and extensive, press upon the coronary arteries and interfere with nutrition in such a manner that the power of the heart becomes insufficient. These are the same symptoms that develop in primary affections of the heart muscle, and in uncompensated valvular lesions, and which we will term stasis symptoms. Palpitation, accelerated irregular pulse, œdema, scanty diuresis, high specific gravity of the urine and slight albuminuria, enlargement of the liver, ascites, hydrothorax, bronchial catarrh, and hemorrhagic infarctions, dyspnoea, and cyanosis—these are the most important features of the clinical history. Exacerbations and remissions alternate frequently until the case terminates fatally. Duroziez recently emphasizes the fact that sudden death may occur (compression and closure of the coronary arteries?).

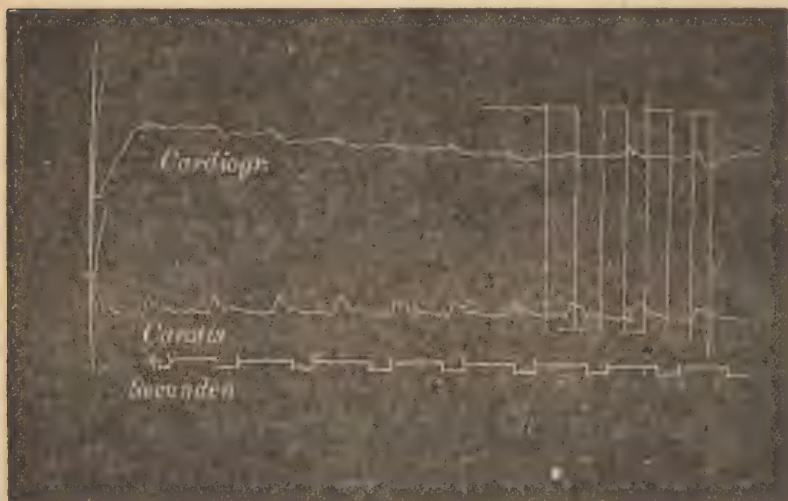
The physical changes are not directly connected with the functional disturbances; either may exist independently. They occur only when the locomotion of the base of the heart downwards is prevented by the pericardial adhesions, although the heart muscle possesses sufficient power. If the heart contracts under such circumstances, and its long diameter diminishes, this can evidently not occur unless the region of the apex beat of the heart is removed from the inner surface of the thorax during systole. But as the anterior border of the left lung is unable to fill the space thus made empty, the part corresponding to the apex beat will be driven inwards during systole by the external atmospheric pressure. The phenomenon will be much more distinct if the contracting heart draws directly upon the inner surface of the thorax on account of adhesions in the neighborhood of the apex. We then find that several intercostal spaces and the lower part of the sternum are pressed inwards at each systole. We see, therefore, that the principal changes are connected with the apex beat, and that they may vary from simple disappearance to extensive systolic retraction. Adhesions which do not prevent movement of the base of the heart will not produce the symptoms described, and, therefore, very extensive synechia may remain without symptoms, while in other cases trifling adhesions give rise to marked systolic retraction of the region of the

apex. It is readily understood that synechiæ of the base of the heart are particularly important in this respect. If extrapericardial adhesions are also present, these symptoms are developed so much more readily because the locomotion of the heart is then restricted usually to the greatest extent, and the phenomenon of systolic retraction must be especially marked when extrapericardial adhesions are present anteriorly at the wall of the thorax and posteriorly at the spinal column at the same time. But as the retraction implies a certain amount of force, the symptom disappears temporarily as soon as the power of the heart muscle fails.

The systolic retractions do not coincide with the beginning of cardiac systole, and v. Dusch has shown that it is a little later than the carotid pulse, but coincides with the radial pulse (vide Fig. 4). Friedreich observed that it increases during inspiration.

Friedreich also called attention to another important symptom of pericardial adhesions, to the diastolic collapse of the cervical veins, *i. e.*, they become very full during systole, and suddenly collapse during diastole. The collapse manifests diastolic mictism, inasmuch as there is first a slight and then a more marked emptying of the veins. The diastolic

FIG. 4.



Apex beat and carotid pulse curve in a case of synechia pericardii. Taken with the aid of Ludwig's kymographion. Personal observation.

collapse of these veins is explained by the fact that the aspiration of the venous blood in the heart is favored by the diastolic projection of the chest walls, and that, in addition, the aspiratory action is increased by the relative diastolic depression of the heart. Under certain circumstances, we may notice systolic projection and diastolic depression of the supra-clavicular region corresponding to the distention and collapse of cervical veins. Sibson states that he observed hepatic pulsation in four cases.

Upon palpation, we can readily determine, by comparison with the radial pulse, whether the retraction at the apex is systolic. The dias-



tolie projection of the thoracic walls is sometimes so vigorous that it has been called diastolic heart beat. On auscultation, also, we often notice a vigorous diastolic impulse against the ear. The existence of anterior extra-pericardial adhesions in addition to the intra-pericardial adhesions is recognized by the fact that the apex beat does not change its position during left lateral decubitus, because the position of the heart is entirely stationary.

Percussion furnishes no direct diagnostic data. But the obliteration of the complementary pleural space or adhesion of the anterior edges of the lungs is recognized by the absence of diminution of the small (absolute) cardiac dulness during inspiration; furthermore, the apex beat is not weakened.

On auscultation, the diastolic projection of the thoracic wall gives rise to a dull sound, which immediately follows the diastolic ventricular sound, so that the latter appears reduplicated. The heart sounds assume occasionally a metallic quality, and become so loud that they are heard at some distance from the patient. This occurs from gastric resonance, and as this requires a certain amount of space, the metallic sound is heard only at times. As the heart and stomach are very close to one another when the pericardium is obliterated, we can readily understand why the heart sounds have a metallic resonance in this condition. But this symptom has no pathognomonic significance.

4. We have previously stated that very many cases of pericardial adhesions present no symptoms, even if the pericardium is entirely obliterated.

We can rarely, from the functional disturbances, diagnose pericardial adhesions with any degree of certainty, as the same symptoms occur in uncompensated valvular lesions and primary disease of the heart muscle. Valvular lesions may be readily excluded if endocardial murmurs are absent, but the question then remains whether we have to deal with a primary muscular affection of the heart or with a secondary affection as the result of pericardial adhesions.

The statement made by Samuel Wilks that symptoms of insufficiency in the power of the heart, in the absence of valvular lesions, is evidence in young persons of pericardial adhesions, in older ones of primary degeneration of the heart muscle, is correct as a general thing, but in a concrete case we cannot go beyond a probable diagnosis.

Caution must also be exercised in employing the physical symptom for purposes of diagnosis. Very little may be inferred from the absence of the apex beat, since this may happen in obesity, pulmonary emphysema, aortic and mitral stenosis, and in conditions of cardiac weakness; at all events, all these conditions must be excluded before the symptom can be attributed to pericardial adhesions. It can only be used in diagnosis if we have followed the course of the disease. It may be attributed to pericardial adhesions if the apex beat could be seen and felt before the development of a pericarditis, and disappeared after the subsidence of the disease.

Systolic retraction, confined to the region of the apex beat, cannot be attributed invariably to pericardial adhesions, as this symptom is also caused by other conditions. Traube showed that it may be the result of abnormal folds on the pericardium. It may also occur in aortic stenosis and, according to Galvagni, in atheroma of the aorta, inasmuch as the locomotion of the heart downwards is hindered on the slight recoil in the first case, and the abnormally slight extension of the arch of the

aorta in the second case. When the anterior border of the left lung is infiltrated and adherent, systolic retractions may also appear, because the edge of the lung cannot follow the systolic contraction of the heart. Furthermore, if the heart has assumed an abnormal position, so that it is turned upon its long axis, and one lateral border is turned anteriorly, the other posteriorly, systolic retraction may result, because the present abnormal diameter shortens, during systole, from before backwards. Systolic retraction in the third and fourth intercostal spaces, close to the left edge of the sternum, has no significance, and occurs in many healthy people. It is found particularly when the intercostal spaces are broad and thin, and the action of the heart is excited.

Consequently, adhesion of the layers of the pericardium can only be diagnosed when the systolic retraction is not confined to the region of the apex beat, but involves a large part of the chest-wall and lower part of the sternum, and when diastolic collapse of the cervical veins is also present.

5. The prognosis is not especially favorable. If the power of locomotion of the heart is obstructed, a part of its action, which would have been employed in the propulsion of the blood, is evidently lost. If the adhesions are of such a character that the nutrition of the heart muscle is disturbed, the rapid development of cardiac insufficiency must be dreaded.

6. Slight adhesions may recover spontaneously. This is shown by autopsies in which a separation of fibrous bands has been observed.

Treatment of the affection is useless, and we must confine ourselves to warning the patient against excessive bodily exertion, to sparing and strengthening the power of the heart as much as possible, to ordering easily digested food, and to combating the threatening symptoms of heart failure by the cautious administration of small doses of digitalis.

#### *b. Mediastino-Pericarditis, Mediastino-Pericarditis fibrosa s. callosa.*

In the discussion of pericarditis, we called attention to the fact that the inflammation may extend to the outer surface of the pericardium, and thence to the connective tissue of the mediastinum. The latter may terminate in fibrous adhesions, either in the form of threads or strands, or membranes and callosities. Remains of thickened purulent or cheesy exudation are also met with. We can easily understand that the large vessels of the heart are readily affected thereby, and undergo flexion, torsion, and immediate or mediate adhesions to the sternum, œsophagus and spinal column. These changes constitute the anatomical picture of

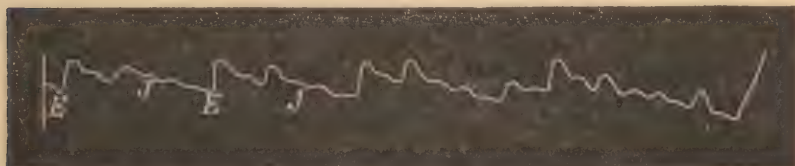
#### *Mediastino-pericarditis.*

Kussmaul first showed in 1873 that this condition is capable of diagnosis in certain cases. The diagnosis depends on the coincidence of two symptoms, *pulsus paradoxus* s. *inspiratio intermittens*, and the inspiratory enlargement of the cervical veins.

Kussmaul describes as *pulsus paradoxus* that form of the pulse which grows smaller during inspiration, and even disappears entirely during deep inspiration. This peculiarity is especially distinct in the graphic tracings of Marey's sphygmograph (vide Kussmaul's original curve in Fig. 5). Kussmaul attributes this pulse to the fact that, during inspiration, the ascending portion and arch of the aorta are drawn upon by the

anterior and posterior fibrous adhesions, so that the outflow of blood from the heart into the arterial system is interfered with. This also

FIG. 5.



Pulsus paradoxus. After Kussmaul. *J*, Inspiration. *E*, Expiration.

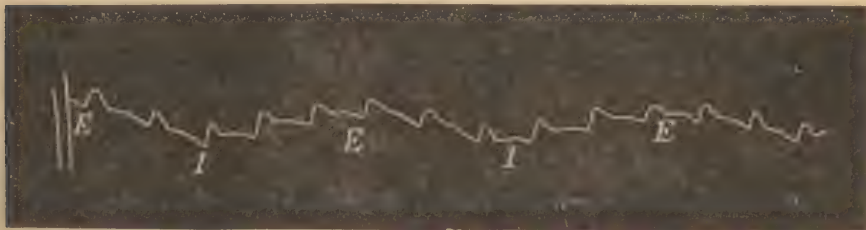
explains the inspiratory dilatation of the cervical veins, as the innominate veins are also bent and narrowed by fibrous adhesions.

But the pulsus paradoxus is not sufficient proof of the existence of callous mediastino-pericarditis, as it is observed also in exudative pericarditis, and Maixner even noticed it in extensive exudation into the left pleural cavity with marked dislocation of the heart towards the right. It disappeared in this case after thoracentesis, but reappeared when the fluid reaccumulated. Maixner also observed the paradox pulse in a case of pyo-pneumo-thorax.

F. Frank observed it frequently in aortic aneurism, and in aneurisms of the large vessels starting from the aorta (but only in the corresponding peripheral distribution of these vessels), and in two cases of patency of the ductus Botalli. Frank explains the first case by the fact that a larger surface of the vessel is exposed to the intra-thoracic pressure. In the two latter cases he assumes that the pressure in the pulmonary artery is diminished during inspiration, so that a portion of the blood passes in larger quantity through the open ductus Botalli from the aorta into the pulmonary artery.

Riegel found that a form of paradox pulse may be recognized in the graphic tracing of the pulse of healthy individuals. The respiratory changes in the pulse are not distinctly perceptible on palpation, although Sommerbrodt states that he has observed that almost complete intermission of the pulse may occur during deep inspiration in healthy individuals. In Fig. 6, it is readily seen that the pulse becomes small

FIG. 6.



Influence of respiration on the pulse curve. Right radial artery. A man, æt. 42 years. Personal observation.

during each inspiration, though it also undergoes other changes. During inspiration the recoil elevation is more distinct, and is lower than during expiration. The elevations of elasticity, however, are less marked



during inspiration than during expiration. The quickness of the pulse is also greater during inspiration than during expiration.

The remarks on pericardial adhesions hold good concerning the termination, prognosis, and treatment of this affection.

## 2. Accumulation of Gas in the Pericardium. *Pneumo-pericardium.*

(*Pneumatosis pericardii et hydro-pneumo-pericardium.*)

I. ETIOLOGY.—Accumulation of gas within the pericardium occurs so rarely that many experienced physicians have never observed it. This condition develops when the pericardium is opened from within or without, and communicates with the external air or an adjacent air-containing organ. As the heart diminishes in volume during each systole, the conditions for aspiration of air are not unfavorable if a valvular fistula is present.

Among the causes of pneumo-pericardium are:

a. External injuries which lay open the pericardium, for example, incised and gunshot wounds. But, as v. Bamberger showed in one case, we must not look upon pneumo-pericardium as an absolutely necessary result of an opening into the pericardium resulting from an injury.

b. Contusion of the thorax, in which the lung and pericardium are laid open on account of fracture of the ribs, or the left lung, which was adherent to the pericardium, and the pericardium itself burst, and permit the entrance of air into the pericardium.

c. Pulmonary cavities, or pyo-pneumo-thorax, when they rupture into the pericardium.

d. Ulcerative processes in the œsophagus, giving rise to an opening into the pericardium, viz., cancer, diphtheritic ulcers, foreign bodies (false teeth, in one case).

e. Ulcerations of the stomach which, after preceding adhesion of the stomach and diaphragm, perforate into the pericardium. This occurs particularly in cancer and ulcer of the stomach. Graves mentions a case in which an hepatic abscess opened into the stomach and pericardium, and gave rise to pneumo-pericardium. Diseases of the other abdominal organs may give rise, in a similar manner, to this condition.

f. More rarely the perforation of a purulent pericarditic exudation into air-containing organs. Meigs described perforation into the œsophagus, and Mueller observed pneumo-pericardium after perforation into the left lung.

Cases of pericarditis also occur in which gas accumulates in the pericardium, although no perforation is found on autopsy. It has been assumed that this is the result of decomposition of the exudation. But this view is not favored by recent analyses, which seem to show that we have to deal with the entrance of air, the fistulous opening being overlooked.

Observations in which gas is said to have developed spontaneously are more than doubtful (probably post-mortem development of gas).

II. ANATOMICAL CHANGES.—If the amount of gas is not too small, we are struck, on opening the thorax, by the marked spheroidal tension of the pericardium. On puncture the gas escapes in part, and with such force as to put out a burning match held in front of it. As a rule the gas is foul-smelling, like sulphuretted hydrogen. Inflammatory changes are always found in the pericardium; if they are absent, we must suspect that the phenomenon is post-mortem in its development. The exudation present is usually purulent, more infrequently hemorrhagic, very

rarely mainly fibrinous; the exudation usually is also ichorous. Mueller has described one case of sero-pneumo-pericardium. As a rule, therefore, there is no pure pneumo-pericardium, but almost always pyo-pneumo-pericardium—a fact which also presents clinical importance. The lower lobe of the left lung is usually compressed to a considerable extent, and the diaphragm is pushed downwards not infrequently on account of the pressure.

All the other anatomical changes depend upon the primary affection.

III. SYMPTOMS.—Physical changes play the chief part among the symptoms. Among them the metallic phenomena alone are dependent on the pneumo-pericardium, while the simultaneous presence of air and fluid in the pericardium gives rise to the striking changes in dullness and the peculiar metallic splashing sounds.

The marked prominence of the cardiac region (voussure) is noticeable in many cases on inspection. The apex-beat is usually not visible, because the heart, which has fallen backwards, is covered anteriorly with fluid and gas; it makes its appearance occasionally when the patient sits erect or is bent over forwards.

On palpation, vocal fremitus is found to be absent over the cardiac region. At times pericardial friction sounds may be felt. Occasionally we notice gurgling, crackling, splashing râles which correspond acoustically to very peculiar sounds.

On percussion the dull sound of the cardiac region has disappeared, and is replaced by a loud, tympanitic note, which also has a metallic quality. The metallic quality is brought out with special distinctness by pleximeter-percussion, in which the pleximeter is percussed with the finger-nail, or with the handle or button of the percussion hammer. The percussion between two hard objects is particularly adapted to bringing out the high notes peculiar to the metallic sound. The distinctness of the metallic quality is also increased by auscultatory percussion, *i. e.*, auscultation of the chest during percussion. Feine observed in one case that the percussion note became lower during cardiac systole, and attributes this to the fact that the heart then approaches the chest-wall and pushes the gas backwards. Gerhardt has also observed that, when the percussion blows follow one another rapidly and uninterruptedly, a change of pitch is occasionally observed with each systole and diastole of the heart. This is owing to the fact that the pericardium, which is filled with gas, undergoes a change during the cardiac movements, and that the tympanitic note over a cavity filled with air is so much lower in pitch, the greater the lumen of the cavity. Some authors state that they have occasionally heard cracked-pot resonance even when there was no open fistula into the pericardium, but it seems to us that this must have been confounded with a very high, distinct, and short metallic note. When the body is bent over forwards, the tympanitic note may be converted into a dull sound, inasmuch as the heart is applied directly to the wall of the chest, and the gas passes backwards.

If, as is the rule, fluid is also present in the pericardium, a tympanitic metallic note in the cardiac region is observed usually only in the dorsal position. In the erect position or in lateral decubitus, the fluid sinks downwards or laterally, and the corresponding parts furnish a zone of dull sound which changes with every position of the body. But this is only possible if there are no adhesions between the two layers of the pericardium, so that the movement of the fluid in the pericardium is unob-

structed. That some authors did not detect any change in pitch may have been the result of pericardial adhesions. Upon assuming the erect position the percussion sometimes becomes higher in pitch, because the greatest diameter of the pericardium is diminished in size by the collection of fluid posteriorly.

Upon auscultation, the heart sounds usually have a striking metallic quality, the result of resonance within the pericardium which is filled with gas. The metallic quality may be so pure that it has been compared to the notes of a zither, or chimes. Occasionally the heart sounds are so accentuated that they may be heard at a little distance from the patient. In some cases, however, they are very faint and dull, perhaps on account of the accumulation of a large amount of fluid in the pericardium, covering the anterior surface of the heart and masking the sounds.

Pericardial friction murmurs are heard occasionally and may also assume a metallic character.

The presence of fluid is shown on auscultation by very peculiar, metallic, splashing, gurgling, rattling sounds, which result evidently from the shaking of the fluid by the movements of the heart. These have been compared with the sound of a revolving mill wheel (*bruit de moulin*, *bruit de roue hydraulique*). They are so loud, occasionally, that they may be heard through several apartments, and Stokes reports a case in which they prevented the patient from sleeping. Even the patient's wife, who slept in the adjoining room, was disturbed by the loud noise.

It has been stated previously that these murmurs may sometimes be felt.

Some authors have described the murmur of falling drops, and a succussion sound on shaking the patient may also be expected.

Eisenlohr observed, in one case, disturbances of deglutition, caused by pressure of the distended pericardium upon the œsophagus.

The subjective phenomena and the other symptoms depend in part on the causes of the disease, in part on the quantity of gas and fluid accumulated in the pericardium. If pneumo-pericardium follows a pericarditis and the quantity of gas is not too large, the disease may present no striking symptoms and may be recognized only on physical examination.

If a large amount of gas accumulates suddenly and exudative pericarditis then develops, symptoms of collapse and heart failure develop, the latter as the result of the increased pressure within the pericardium. The patients complain of a feeling of oppression and dyspnoea, occasionally of stitches in the cardiac region; the pulse is accelerated, small and often irregular; livid color of the skin; later more marked symptoms of stasis, followed by death. Death is preceded occasionally by delirium and convulsions.

IV. DIAGNOSIS.—The disease is easily recognized. The diagnosis is based on disappearance of cardiac dulness, change of percussion sound in the cardiac region in different positions of the body, metallic splashing sounds, and, in general, phenomena which present a metallic quality.

Diagnostic errors are conceivable if cavities, containing air and fluid, are adjacent to the heart. Under such circumstances the heart sounds may have a metallic quality from resonance in the cavities, and when the air contained therein is shaken, the development of metallic splashing noises is possible. This may occur in large pulmonary cavities situated near the heart, in encapsulated pyo-pneumo-thorax, and in marked dis-



tention of the stomach when it is filled partly with fluid. All these conditions are distinguished from pneumo-pericardium by the presence of cardiac dulness, although this may be displaced laterally by pneumothorax.

V. PROGNOSIS.—The prognosis is unfavorable. Among fourteen cases collected by Friedreich, ten died (71.3 per cent). Death occurs generally between the first and twelfth days. Recovery can be looked for only in cases of traumatic pneumo-pericardium in which neither infecting air nor foreign bodies enter the pericardial cavity. At times a pyo-pneumo-pericardium may be converted into a pyo-pericardium by gradual absorption of the gas. In many cases the prognosis is rendered unfavorable by the primary affection itself.

VI. TREATMENT.—In traumatic pneumo-pericardium the inflammation should be combated by the application of an ice bag to the cardiac region; if symptoms of collapse are present, wine and stimulants are indicated. Marked feeling of oppression and insomnia should be treated with narcotics, preferably by injections of morphine. If the heart's action is very much accelerated, digitalis may be cautiously administered.

The same therapeutic principles also hold good in other forms of pneumo-pericardium.

If the tension of the gas seems excessive, the pericardium should be punctured, and the gas then carefully and gradually aspirated. But if profuse purulent or ichorous exudation is present, it seems advantageous to open the pericardium under strict antiseptic precautions, and then to act according to the principles of modern surgery. As a matter of course, this should be done only when the primary affection does not appear hopeless.

### 3. Dropsy of the Pericardium.

(*Hydropericardium, Hydrocardie.*)

I. ANATOMY.—Clear serous fluid (liquor pericardii) is found within the pericardium of nearly all corpses. Some writers believe that the pericardium contains a small quantity even during life, while others maintain that the liquor pericardii is formed during the death agony or even after death. The latter view is in conformity with the experience that the amount of the fluid is so much larger, the longer the death agony has lasted and the longer the autopsy has been delayed. v. Luschka first maintained that the liquor pericardii is produced mainly by transudation from the thin-walled right auricle, because this part of the heart is always found distended with blood at the autopsy. The quasi normal amount of liquor pericardii averages 5-10 ccm. It may also amount to 100-120 ccm., without justifying us in assuming a morbid process. Beyond this amount we are justified in assuming a pathological condition, known as hydrops pericardii.

It is peculiar to dropsy of the pericardium that inflammatory processes are not involved directly in the genesis of the fluid, so that it is readily differentiated from pericarditis. The dropsical fluid is clear, amber yellow, occasionally greenish, and usually presents dichroism, inasmuch as it is yellow by transmitted light, and emerald green in reflected light. It is occasionally slightly reddened from mixture with the coloring matter of the blood. This occurs sometimes post mortem

from imbibition; more rarely blood-globules pass into the fluid during life, and after the transformation of their coloring matter, impart to the fluid a dirty brownish color. This is observed particularly in cancer and tuberculosis of the pericardium. In jaundice, the fluid presents a golden yellow, icteric color, and bile pigment and biliary acids can be demonstrated in it.

Not infrequently it contains small, delicate gray flocculi, consisting of swollen, granular or fatty desquamated endothelium cells of the surface of the pericardium. In several cases I have observed small glistening crystals, which proved under the microscope to be tablets of cholesterolin. The fluid is extremely poor in cellular constituents, though now and then a swollen, fatty round cell is met with.

There is often a grayish-yellow gelatinous exudation of fibrin which is deposited either in the form of small threads and flocculi, or of large coherent masses.

The fluid is always alkaline. Urea and sugar have been found in it. According to the analyses of v. Gorup-Besanez (I.), Wachsmuth (II.), and Hoppe-Seyler (III.), it consists of :

	I.	II.	III.
Water, . . . . .	955.1	962.5	961.78
Solid constituents, . . . . .	44.9	37.5	38.22
Fibrin, . . . . .	0.8	..	..
Albumin, . . . . .	24.7	22.8	24.63
Extractive matters, . . . . .	12.7	..	..
Inorganic salts, . . . . .	6.7	..	..

The quantity of fluid may amount to 1,000 ccm. or more, and Corvisart states that he has seen an accumulation of 4,000 ccm.

If the amount of fluid is not too small, the pericardium presents a striking appearance, even before it is opened, by its tension and fluctuation. After it is opened, the surface appears smooth, shining, and free from inflammatory changes and opacities. It usually looks very pale on account of the compression of the pericardial vessels. As a result of the great tension the pericardium is not infrequently thinned, though thickening of the parietal layer has also been described. The subepicardial fatty tissue has often disappeared, and the heart muscle is occasionally pale, dull, slightly macerated or fatty in appearance. The right ventricle is usually dilated, as a result of the causes underlying the pericardial dropsy. The subserous connective tissue is sometimes œdematous, and the œdema may spread even to the adventitia of the large vascular trunks.

(Edema is also found occasionally in the cellular tissue connecting the pericardium with the mediastinum and diaphragm.

The lower lobe of the left lung is often compressed, and the diaphragm is not infrequently depressed.

All other changes depend on the primary disease.

II. ETIOLOGY.—Pericardial dropsy is never a primary, independent disease, but is always dependent on circulatory disturbances or on conditions of diminution of albumin in the blood. These causes are the same as those of other dropsies, thus explaining the fact that the disease is associated almost always with œdema of the skin, ascites, hydrothorax, etc.

Among the circulatory disturbances, chronic pulmonary affections, diseases of the pleura, valvular affections, degeneration of the heart muscle, and spinal curvatures are specially important. All these condi-

tions may obstruct the outflow of blood from the pulmonary artery, so that the stasis is propagated into the right ventricle, the right auricle, and then into the venæ cavae, and also into the coronary veins and through these into the pericardial veins.

Much more rarely local stasis occurs in the coronary veins alone—cases in which the hydropericardium appears to be a primary affection. This is observed in cancer and tuberculosis of the pericardium when individual tumors compress the coronary veins; calcification of the coronary arteries may exercise a similar effect. New growths in the mediastinum and retracting fibrous bands are said to have the same effect.

Hydropericardium, as the result of diminution of albumin in the blood, occurs, apart from Bright's disease, in all cachectic conditions (cancer, tuberculosis, chronic diarrhœa, protracted loss of blood, malaria, etc.).

A few writers, and among them even v. Bamberger, have assumed the existence of hydropericardium *ex vacuo*. This is said to occur in atrophy of the heart and retracting pulmonary and pleural processes, because the fluid, to a certain extent, acts as a filling mass for the empty space which has resulted. To say the least, the existence of this form of hydropericardium is unproven, and it is not very probable, since the diaphragm, lungs, and chest-walls could more readily fill the assumed empty space.

III. SYMPTOMS.—Hydropericardium can only be recognized with certainty during life by the aid of physical exploration, and then only when the quantity of fluid is not too small. All the symptoms are then produced which depend on the presence of fluid in the pericardium, and which were discussed *a propos* of pericarditis. As a matter of course, a friction murmur, which is indicative of inflammatory processes in almost every case, will not be present.

The following are the symptoms in question: *a. Inspection*.—Projection of the cardiac region; dilatation of the intercostal spaces; elevation of the left nipple; disappearance of the apex beat, which appears occasionally only in the erect position, when the body is bent over forwards. *b. Palpation*.—Absence of vocal fremitus in the cardiac region. *c. Percussion*.—Trapezoidal form and enlargement of cardiac dulness; increased height of cardiac dulness in the erect position; cardiac dulness extending to the left beyond the apex beat; the cardiac dulness of unusual extent inferiorly and superiorly. *d. Auscultation*.—Muffled heart sounds.

In addition there may be signs of compression of the lower lobe of the left lung (dulness, increased vocal fremitus and bronchial breathing).

We will not discuss the symptoms dependent on the primary affection.

Subjective symptoms may be entirely absent, though many patients complain of a peculiar feeling of tension and pressure in the region of the heart. It is a peculiar fact that a considerable accumulation of transudation may disturb the action of the heart only to a slight extent, while inflammatory fluids cause great disturbance when present even in much smaller quantities. In the latter condition, therefore, it is evident that a part of the symptoms of insufficient action of the heart is owing to an inflammatory implication of the heart muscle itself.

The objective symptoms of hydropericardium, apart from those mentioned, consist of symptoms of stasis; but it must be remembered that these are caused, as a rule, by the primary affection. The symptoms in



question are cyanosis, enlargement of the veins, anasarca, ascites, enlargement of the liver, diminished diuresis, occasionally albuminuria, hydrothorax, bronchial catarrh, or hemorrhagic infarctions, and signs of cerebral hyperamia (somnolence, delirium, even convulsions).

The patients are often distressed by dyspnoea and a feeling of oppression; the pulse is small, very frequent, and irregular in rhythm and vigor.

IV. DIAGNOSIS.—The recognition of hydropericardium is easy if the fluid is sufficient in amount to give rise to changes in cardiac dulness. For the differentiation of cardiac dulness from affections of the heart muscle, lungs, pleura and mediastinum, vide the chapter on pericarditis (page 15).

When the presence of fluid is demonstrated, we must decide between hydropericardium and pericarditis. The absence of fever and pain in the cardiac region, the presence of other varieties of oedema, and the consideration of the etiology, will render certain the diagnosis of dropsy of the pericardium.

V. PROGNOSIS.—The prognosis is unfavorable in many cases on account of the primary affection. Hydropericardium, as such, is attended much less frequently with danger to life. Complete absorption of the fluid and recovery are possible, but sometimes partial absorption and re-accumulation of the fluid alternate with one another.

VI. TREATMENT.—The treatment of hydropericardium is almost always the same as that of the primary affection, and according to circumstances, therefore, we must administer diuretics, laxatives, diaphoretics, and roborants. In addition, due weight must be attached to proper diet.

Not much benefit may be expected from the local application of tincture of iodine, ointments of iodide of potassium or iodoform. In this disease, as in pericarditis, v. Bamberger recommends repeated blistering.

Puncture of the pericardium is indicated when the quantity of fluid in the pericardium is so large that symptoms of insufficiency of the heart's action must be attributed to excessive compression of the heart muscle.

#### 4. *Accumulation of Blood in the Pericardium. Hæmopericardium.*

An accumulation of blood in the pericardium, independent of inflammatory changes, is rarely observed. It is found in injuries of the pericardium and heart muscle, in rupture of aneurisms of the coronary arteries, aorta, and pulmonary artery, in rupture of the heart as the result of fatty degeneration, abscess, and acute and chronic aneurism of the heart.

The quantity of blood effused may be very considerable, and exceed 500 ccm. In such cases, the pericardium is distended, and often allows us to suspect its bloody contents by its bluish-black color. The amount of blood is larger, as a rule, when the blood trickles slowly into the pericardium, and gradually distends it, than when the hemorrhage occurs suddenly. In the latter event, the pericardial cavity is soon filled, and thus causes stoppage of the hemorrhage (spontaneous tampon). The blood is partly fluid, partly clotted. If death occurred rapidly, other changes in the pericardium are absent; otherwise inflammatory processes soon develop.

The diagnosis depends, in the first place, on the demonstration of fluid in the pericardium. Next are the signs of internal hemorrhage: pallor and coldness of the skin, disappearance of the pulse, vomiting, ringing in the ears, spots before the eyes, vertigo, loss of consciousness, convulsions (from cerebral anemia). These symptoms may lead at once to a fatal termination. Increasing anemia develops gradually in those cases in which the extravasation occurs slowly. On the other hand, death may occur suddenly and without premonition, as has been observed particularly in extensive rupture of the heart.

The prognosis is unfavorable on account of the causes of the disease, and recovery is possible only in traumatic hæmopericardium.

Treatment consists of the application of an ice-bag to the cardiac region, the use of stimulants and roborants, in certain cases of styptics, particularly subcutaneous injections of ergotin (ergotinum Bombellon, one-half syringe-ful mixed with an equal quantity of water). I recently examined a case of traumatic hæmopericardium (pistol shot wound) in which puncture of the pericardium, followed by aspiration, was attended with brilliant results.

### 5. Tumors, Parasites, and Free Bodies of the Pericardium.

*a. Tubercle.*—During general miliary tuberculosis, miliary and sub-miliary gray nodules also develop on the pericardium, though this serous membrane does not present any pronounced predisposition to the affection. The miliary tubercles are usually most abundant on the epicardium, particularly in the vicinity of the vessels.

In very rare cases tuberculosis of the pericardium is primary, *i. e.*, independent of cheesy and tubercular processes in other organs. In a case described by me the cheesy tubercle had given rise to large undermined ulcers, similar in appearance to tubercular ulcers of the intestines, and such a profuse hemorrhage occurred that death followed in a few hours. A similar termination, though not in primary tuberculosis, is mentioned by Riegel.

Sometimes tuberculosis of the pericardium develops after a previous pericarditis, the cheesy remains of the exudation becoming infected with tubercle bacilli. The gray or cheesy nodules are then found particularly between the two layers of the pericardium, in the fibrous adhesions which have developed as the result of the inflammation.

As a rule, tuberculosis of the pericardium is the result of and associated with cheesy tubercular processes in other organs, particularly the lungs. Occasionally only that portion of the external layer of the pericardium is covered with tubercles, which is situated next to a tubercular focus in the lungs, mediastinum, or beneath the diaphragm (local infection).

This is associated frequently with secondary pericarditis, which often has a hemorrhagic character and chronic course. In such cases the pericardium is not infrequently callous and more than 1 cm. in thickness, and its surface strewn with large cheesy nodules, and with cheesy and gray transparent tubercles. It has been previously mentioned that, in addition to inflammatory exudations, dropsical accumulations in the pericardium may also take place.

A positive diagnosis of these conditions is not possible. A probable diagnosis may be made when signs of effusion in the pericardium appear in an individual suffering from tuberculosis and caseation of other

organs, but mistakes are possible because non-tubercular pericarditis may also develop under such circumstances.

*b. Cancer and Sarcoma.*—Cancer and sarcoma of the pericardium develop most frequently from direct propagation from adjacent organs (cancer of the œsophagus, sternum, mammary glands, external integument, and sarcoma of the mediastinal lymphatic glands or the thymus gland). These neoplasms are observed more rarely as true metastases *i. e.*, in similar affections of remote organs. Foerster described a case of primary cancer of the pericardium.

In some cases there is a diffuse cancerous or sarcomatous infiltration, in others there are isolated or confluent nodules. The tumors are usually succulent, medullary, rarely dry canceroids. In addition there are usually inflammatory or dropsical effusions, the former usually of an hemorrhagic character.

*c. Gummata* in fibrous adhesions of the pericardium are mentioned by Lancereaux and Orth.

*d. Parasites.*—Free trichinæ have been found occasionally in the pericardium. Cysticerci are sometimes situated in the subepicardial fatty tissue. Echinococci also occur, occasionally as free vesicles in the pericardial cavity.

*e. Free Bodies.*—These have been found a number of times in the pericardium. They are derived generally from constricted portions of a neoplasm or inflammatory connective tissue proliferations, which may, at times, undergo calcification. Hyrtl describes a case in which the concretion, which was freely movable within the pericardium, consisted of a calcified bronchial gland, which had perforated into the pericardium. Foreign bodies (needles, nails, etc.) are rarely observed in the pericardial cavity. A diagnosis is impossible.

### 6. *Milk Spots of the Pericardium. Maculæ Tendinæ.*

Milk spots are white, tendinous thickened patches on the pericardium, found usually on the visceral, more rarely on the parietal layer. They occur so frequently in advanced age that many authors deny their pathological significance. They are observed very rarely in the young, but Hodgkin found them in a child of ten weeks. They seem to be somewhat more frequent in men than in women.

They are situated almost constantly on the anterior surface of the right ventricle, immediately beneath the beginning of the pulmonary artery. They are also found not infrequently on the anterior surface of the left ventricle, a little above the apex; also along the coronary arteries. They are much more rare upon the posterior surface over the auricles and near the origin of the great vessels. Milk patches are present occasionally in several places at the same time.

Their size varies from that of a pea to that of a five-mark piece. Sometimes they are rounded, sometimes more elongated or stellate, and are either sharply defined or merge gradually into surrounding parts. Their surface is usually smooth and the endothelium is continued uninterruptedly over them. More rarely they present a corrugated, rough and villous surface. Sometimes they are swollen like gelatin by œdema.

On microscopical examination they are found to constitute a hyperplasia of the serous and occasionally the subserous connective tissue.

Opinions vary with regard to their causes. A small proportion result from inflammatory changes in the pericardium. The majority are non-



inflammatory and arise from mechanical irritation received by the pericardium in the movements of the heart against the inner surface of the thorax, and must therefore be placed in the same category with callosities of the skin. This view accords with the fact that the milk patches often develop in those places at which the heart is uncovered by the lungs and is applied directly to the thoracic walls. It is also corroborated by the fact that in cirrhosis of the liver Hodgkin observe milk patches on that portion of the heart's surface which was turned towards the rough surface of the liver. If all milk patches were attributed to inflammatory changes, it would be inconceivable that pericarditis should occur so often without any symptoms whatever.

These changes produce no symptoms during life and therefore possess only an anatomical interest. The view of some writers that the patches may give rise to a pericardial friction murmur is contradicted by very experienced clinicians.

### 7. *Defects and Diverticula of the Pericardium.*

*a. Defects.*—Congenital defects of the pericardium have been described several times (compare Faber, Virchow's *Archiv*, Bd. 74). Sometimes there are abnormal openings and fissures in the pericardium, sometimes the parietal layer is almost entirely absent, so that only a few rudiments may hang like fringes from the origin of the great vessels. In the latter event the heart lies either between both pleural sacs or it is drawn completely into the left pleural cavity and receives from the left pleura a serous covering formed by involution. Such changes may be unattended with any disturbance and often are only observed accidentally at the autopsy.

In Baillie's case the heart had assumed an abnormal position, the apex upwards, the base downwards. This patient was a female infant which died immediately after birth.

The pericardium is generally absent when the heart is situated outside of the thorax (*ectopia cordis*).

In rare cases defects of the pericardium may result from injury. Baker describes a case of *hernia diaphragmatica*, in which a large part of the omentum was situated in the pericardial cavity. An old cicatrix indicated that the diaphragm and pericardium must have been injured at the same time.

*b. Diverticula.*—Hernial protrusions of the pericardium may be congenital or acquired. The latter develops from pressure from within, or traction from without. In the former the fibrous layer of the pericardium is thinned and pressed asunder so that the serous portion projects through the fissure. The size of the diverticulum varies, but it may exceed the dimensions of a hen's egg. The opening is usually small so that the diverticulum possesses a sort of neck. It is usually filled with fluid and, if sufficiently large, physical signs are possible. Otherwise it presents no clinical interest.

## PART II.

## DISEASES OF THE HEART MUSCLE.

1. *Dilatation of the Heart.*

1. *Etiology.*—Abnormal dilatation may affect all the cavities of the heart or be confined to individual ones. Circumscribed dilatation (cardiac aneurisms) will be discussed later.

It is evident that a cavity of the heart will have a so much greater tendency to dilatation, the less its muscular tissue is able to resist abnormal obstructions. For this reason dilatation is found most frequently and most marked in the thin-walled auricles, and the right ventricle dilates more readily than the thick-walled left ventricle.

The causes of cardiac dilatation are either mechanical (circulatory disturbances), or nutritive (changes in the heart muscle); occasionally both are combined.

Dilatation from mechanical causes always occurs when the pressure upon the inner surface of the auricles or ventricles is increased during diastole. This is most frequent in valvular affections. Although the finer physical process may vary, the final result is always the same. We will take as an illustration the physical changes in insufficiency of the aortic and the mitral valves.

If the aortic valves are insufficient, a part of the blood in the aorta flows back into the left ventricle at the beginning of diastole of the heart, the left ventricle thus being supplied with the normal amount of blood from the left auricle, and also with the regurgitated blood from the aorta. As a matter of course, it cannot accommodate this increased amount of blood, except by increasing in size; indeed, the increase in size must correspond exactly with the insufficiency.

The conditions are somewhat different in mitral insufficiency, so far as regards dilatation of the right ventricle. In mitral insufficiency a part of the blood of the left ventricle regurgitates into the left auricle during systole of the left ventricle. The left auricle must dilate because it receives the regurgitated blood in addition to the blood from the pulmonary veins. The abnormal increase of blood pressure, which is thus produced in the left auricle, is propagated by means of the pulmonary veins, capillaries, and arteries to the right ventricle, and thus furnishes the conditions for the dilatation of the latter.

It is readily seen that dilatation of the cavities of the heart cannot be the sole result of a valvular affection if the circulation of the blood is to continue. For the overcoming of the increased blood pressure and propulsion of the increased mass of blood are only possible if the heart muscle hypertrophies and thus exerts greater force. In this way is explained the fact that dilatation as the result of valvular disease rarely occurs alone, but is associated with hypertrophy of the heart muscle. These two processes, which are termed compensation of a valvular affection, open certain natural sources which seek to eliminate as far as possible the circulatory disturbances produced by the valvular disease. We can also understand that a certain relation will develop between the

dilatation and hypertrophy of the heart, as it is unnecessary to say that the one process follows immediately on the other. If the hypertrophy does not develop, as is observed in valvular diseases occurring in enfeebled, old, or very sick individuals, or if the dilatation assumes the upper hand in previously compensated valvular affections on account of degeneration of the heart muscle, serious disturbances of circulation result, which are manifested chiefly by stasis of the venous system, and will be discussed later in detail.

The necessary results of a valvular affection can be predicted with mathematical certainty. In aortic affections the left ventricle assumes the compensation, in affections of the mitral and pulmonary valves the compensatory aid of the right ventricle is called upon. The right auricle takes this part in tricuspid lesions. So long as only one valve is implicated, the dilatation and hypertrophy are usually present only in single parts of the heart. But if combined valvular affections are present, the entire heart passes, under certain conditions, into a dilated hypertrophic condition, for example, in the combination of mitral and aortic disease. The heart may then assume very unusual dimensions (*cor bovinum*).

The preceding considerations enable us to understand that dilatation of the left or right ventricle must be caused by all elevations of blood pressure in the domain of the aorta or pulmonary artery. Here also, if circulatory disturbances are to be avoided, hypertrophic conditions must develop, and it is thus characteristic to a certain extent of mechanical dilatation of the heart, that it is not isolated, but is associated with hypertrophy of the corresponding portion of the heart.

The most frequent causes of increased blood pressure in the domain of the aorta, are aortic aneurism, stenosis of the trunk of the aorta, arteriosclerosis, contracted kidneys, pregnancy. Elevation of the pressure in the pulmonary artery is found particularly in chronic diseases of the pulmonary parenchyma and pleura, and in spinal curvatures.

Nutritive dilatation of the heart is associated with changes in the heart muscle. It arises from diminished power of resistance of the heart, so that the normal blood pressure is sufficient to dilate the cavities of the organ. Such conditions are often temporary and can be relieved by general invigoration of the constitution. From an anatomical standpoint these conditions vary greatly; in some cases no change can be demonstrated, in others there are grave lesions of the muscular tissue.

Nutritive dilatation is found not infrequently in febrile conditions, because the tonus of the muscular tissue suffers under the influence of the elevated temperature. The change affects chiefly or exclusively the right heart and develops even when cloudy swelling or fatty degeneration of the heart muscle cannot be recognized.

Dilatation also occurs in the course of infectious diseases (typhoid fever, cholera, articular rheumatism, pneumonia, variola, scarlatina, erysipelas, diphtheria, etc.). It is evident that some influence is exerted in this direction by the general enfeeblement of the constitution, because these conditions also develop when febrile symptoms are slight or absent. A similar effect is exercised by other exhausting conditions: repeated losses of blood, protracted digestive disturbances, above all, chlorosis, but also long-standing disease in general. Cardiac dilatation has been observed also in poisoning with alkalis and mineral acids.

According to some authors, excessive bodily exercise also gives rise to acute dilatation of the heart. Thompson reports concerning a man,



at. 28 years, who was seized with severe pains about the heart immediately after lifting a heavy load, soon presented signs of cardiac insufficiency and at the same time considerable increase in the size of the heart, and died within two weeks. Upon autopsy the heart was found remarkably dilated, extremely flabby and fatty.

Among primary diseases of the heart muscle dilatation occurs in fatty degeneration and inflammatory changes of the heart. Stenosis of the coronary arteries and veins may also impair the nutrition of the heart to such an extent that dilatation results. This is observed, among other conditions, in obliteration of the pericardial cavity. Dilatation is also observed not infrequently in pericarditis when the heart muscle has taken part in the inflammation and undergone imbibition of serum. Whether purely nervous disturbances may cause cardiac dilatation is still doubtful. Potain has recently emphasized the opinion that dilatation of the right ventricle develops not infrequently in gastric and hepatic diseases, and Stokes long since maintained that this holds good of many affections of the abdominal organs.

We will devote our attention here solely to the nutritive form of cardiac dilatation.

II. *Anatomical Changes.*—Pure cardiac dilatation, unaccompanied by hypertrophic changes, is characterized by enlargement of individual parts of the heart or of the entire organ, and by thinning of its walls.

When only certain portions are dilated, the heart assumes an unusual shape; so that if one ventricle is dilated, the unchanged ventricle looks like an annex of the dilated one. In dilatation of both ventricles the heart usually loses its triangular shape, becomes more rounded, and is said to resemble a game bag. Upon laying open the heart, the muscle is not alone found thinned, but is also very flabby, and while the healthy heart muscle gapes at the place of incision, the dilated walls collapse. The unusual flabbiness of the muscular substance is often shown by the fact that upon pressure with the finger the muscle folds in like the finger of a glove. If the heart, after the origin of the great vessels has been cut through, is raised by the apex, the flabby muscle sometimes falls like a cap on both sides of the hand.

The thinning of the walls may reach an extraordinarily high grade. The muscular fibres of the auricles may be separated more and more from one another until finally the endocardium and pericardium are almost in contact. In the ventricles the thinning is most striking near the apex, and here, also, the muscular tissue may disappear almost entirely, so that only a small quantity of adipose tissue separates the endocardium from the pericardium. Extreme dilatation and thinning of the trabeculæ are also observed not infrequently, so that they seem to be converted into flat, pale, tendon-like strands.

The heart muscle, as a rule, is extremely pale, and is also often strikingly brittle. Other structural changes may be entirely absent, as is seen not infrequently in febrile diseases. In other cases the microscope shows parenchymatous cloudiness and fatty degeneration, occasionally waxy-like degeneration. Sometimes macroscopical changes are recognized, which depend on fatty degeneration or connective-tissue bands.

The dilated cavities usually contain a large quantity of blood, and signs of venous hyperæmia are generally noticed in the other organs.

The condition in question may readily be mistaken for post-mortem dilatation of the heart. The latter is noticed particularly in individuals who have had a long death struggle, and have suffered from diseases of

the respiratory tract; or who died with symptoms of suffocation. It affects chiefly, if not exclusively, the right auricle and ventricle, and finds its explanation in the fact that the right side of the heart is over-filled with blood towards the end of life. If decomposition sets in, the walls of the heart also may become very flaccid and favor the mistake for vital dilatation of the organ. It should be kept in mind that in post-mortem dilatation the size of the heart diminishes, and not infrequently becomes normal when the clots contained therein are removed. Post-mortem flaccidity is also often recognized by the staining of the internal wall with the coloring matter of the blood.

III. SYMPTOMS.—Among the symptoms of dilatation of the heart the chief place is assumed by the physical changes. Dilatation of the left ventricle is recognized by the fact that the large (relative) cardiac dulness assumes unusual dimensions to the left, and often also inferiorly, inasmuch as it passes beyond the left mammary line, and also extends to the sixth and seventh left intercostal spaces. The apex-beat is not infrequently diffused, but has remarkably little force in contrast to hypertrophy of the left ventricle. The small, soft radial pulse also testifies against hypertrophy.

In dilatation of the right ventricle there is abnormal extension of relative cardiac dulness, and cardiac resistance to the right, the former passing beyond the right border of the sternum, the latter extending more than 2 Cm. beyond it at the level of the fourth costal cartilage.

The cardiac dulness assumes very unusual dimensions towards the right side when there is dilatation of the right auricle, while dilatation of the left auricle gives rise to no objective symptoms, because it is situated so far posteriorly, and is everywhere covered with thick layers of lung.

The heart sounds are characterized often by their slight intensity—an effect of the diminished power of the excessively dilated heart muscle. Occasionally some of the heart sounds are entirely absent. Not infrequently we find systolic murmurs, which must undoubtedly be regarded as accidental. Although certain writers believe that the dilatation may be so great that an otherwise normal valve is insufficient to completely close the valvular orifice, it must be remembered that recent measurements have shown that this requires unusually extensive dilatation, and that, in addition, the further consequence of so-called relative insufficiency, viz.: hypertrophy of the cardiac muscle, remains absent, even if the condition has lasted a long time. In cases of dilatation of the right ventricle from gastric and hepatic diseases, Potain found galloping rhythm (*bruit de galop*), *i. e.*, reduplication of the systolic heart sound over the lower part of the sternum, corresponding to the edge of the right ventricle.

In many cases there is unusual acceleration of the heart's action, intensified occasionally into attacks of palpitation associated with anxiety and dyspnoea, more rarely with pain.

As a rule, signs of venous stasis develop in a short time. The jugular veins are immoderately filled, and often present undulatory and even pulsating movements. Cyanosis is also present. If these changes continue for some time, they may result in œdema, diminution of diuresis, albuminuria, enlargement of the liver, bronchial catarrh, infarctions, and other symptoms of stasis, and finally in death.

The arteries, on the other hand, contain an abnormally small amount of blood, so that the pulse is small and soft even if the left ventricle is

not directly changed. This explains the great tendency to syncope as the result of cerebral anæmia.

The duration of the affection depends upon its causes. Dilatations in consequence of febrile conditions disappear most rapidly, often within twenty-four hours.

IV. DIAGNOSIS.—The disease is easily recognized if we take into consideration the extent of cardiac dulness. Mistakes are conceivable if the heart attains unusual dimensions in all its parts, so that a suspicion of pericarditis is aroused. The true condition is shown by the absence of friction murmurs and the dulness characteristic of pericarditis, and by the characteristic relation of the apex beat to the cardiac dulness. Differentiation from enlargement of the cardiac dulness as the result of infiltration of the anterior borders of the lungs or encapsulated pleuritic exudations is effected, as we have shown in discussing the diagnosis of pericarditis, by the usually irregular course of the dulness, the presence of bronchial breathing and tinkling râles, and by the history of the disease.

V. PROGNOSIS.—The prognosis depends on the primary disease, and is especially grave when extensive changes may be assumed to exist in the heart muscle. In such cases, the disease is usually irreparable, while it is capable, under other circumstances, of complete resolution. At all times, however, it must be regarded as a grave event, since the circulation of the blood necessarily suffers, and adds a serious complication to the primary disease.

VI. TREATMENT.—The treatment depends in the main upon the causation. In chlorotic and exhausting conditions, preparations of iron, roborants, and nourishing diet are indicated, particularly an abundant milk diet. In febrile conditions, we should endeavor to reduce the temperature as rapidly as possible by means of cold baths, the administration of large doses of alcoholics and antipyretics.

In many cases, we must seek to strengthen the heart's action and diminish the number of its contractions by nourishing diet, the application of an ice bag to the region of the heart, and the careful use of digitalis. Stimulants may also be employed under certain circumstances. Papillaud and Cheval state that they have observed increased vigor of the heart and regulation of its innervation from the administration of arsenic preparations and antimony, but these remedies should be given, if at all, with extreme caution. We should, in particular, avoid giving tartar emetic, on account of its tendency to produce collapse. Nor have we seen any but disagreeable effects from the use of veratrine, which is recommended by some.

As a matter of course, the body should always be kept as quiet as possible, in order that the circulation may be spared every unnecessary elevation of blood pressure.

## 2. *Hypertrophy of the Heart.*

### (*Hypersarcosis cordis.*)

1. ANATOMICAL CHANGES.—Hypertrophy of the heart is an increase of the muscular tissue of the organ. It may develop in two ways: either by an increase in the size of each individual muscular fibre (hypertrophy in the stricter sense), or by an increase in the number of



the fibres (hyperplasia). As a matter of course, a combination of both conditions cannot be excluded.

The opinions concerning the tissue-changes in cardiac hypertrophy are still divided. Hepp, Wedl, and others have expressed themselves in favor of hypertrophy, Rindfleisch in favor of hyperplasia of the fibres. Letalle maintains that the changes in the muscular fibres appear in scattered groups, whose distribution is associated neither with the arrangement of the blood-vessels nor with the grouping of the secondary bundles of fibres. The width of the individual muscular fibres may increase to fifteen times the normal dimensions, but does not go beyond 0.31-0.33 mm. Proliferation of the nuclei does not occur, although (perhaps as the result of an irritative process within the fibres) they undergo a change of shape (enlargement, according to Aufrecht).

Not infrequently, there is increase of the intermuscular connective tissue, and Lee and Cloetta have observed thickening of the nerves, though it is doubtful whether the latter depends on increase of the connective tissue or hyperplasia of the individual nerve fibres.

Formerly, the terms true and false hypertrophy were employed. The latter term was applied to thickenings of the muscular wall of the heart, produced by connective-tissue bands, new growths, and the like. Such changes will not be considered in the following remarks.

An hypertrophic heart is characterized above all by abnormal thickness of the walls of the organ.

As dilatation is almost always associated with hypertrophy, there ensues at the same time a very considerable increase in the volume of the heart, such as is not often seen in simple dilatation. The combination of dilatation and hypertrophy of the heart is known as eccentric hypertrophy. The term concentric hypertrophy is applied to that condition in which not alone is no dilatation of the cavities present, but the latter appear smaller on account of the hypertrophy of the muscle. It is said that this diminution may be so great that the cavity of the left ventricle scarcely suffices for the introduction of the little finger. Many authors deny entirely the occurrence of concentric hypertrophy; they regard it as a post-mortem phenomenon, resulting from the persistence of the heart in systolic contraction. This appears to be straining a point, as very experienced physicians (for example, Rokitsansky and v. Bamberger) acknowledge its occurrence in rare cases. It has been described a number of times, particularly in combined mitral and aortic disease.

At all events, we must be on our guard against interpreting every thickening of the heart-muscle associated with diminished size of the cavities as concentric hypertrophy. This is observed as a post-mortem change in individuals who have died from hemorrhage, those who have died suddenly from a fall, and in those who have suffered from exhausting discharges, for example, in cholera. Cruveilhier states that it may be distinguished from vital changes by the fact that the apparent hypertrophy can be readily made to disappear upon pressure made by the hand placed within the cavities of the heart.

In simple hypertrophy, the muscular wall of the heart appears thickened, but the dimensions of the cavities of the organ are unchanged.

According to the extent of the hypertrophic changes, we distinguish total, partial, and circumscribed hypertrophy of the heart. In total hypertrophy, all parts of the heart, in partial hypertrophy, only individual parts are affected; in the circumscribed form, the change may be

restricted to portions of a single division of the organ. Thus, certain papillary muscles are occasionally strikingly hypertrophied, or the increase in thickness may be restricted to the septum ventriculorum. In the auricles, hypertrophy is sometimes observed exclusively, or almost exclusively, in the auricular appendages, and in the right ventricle, the *conus anterior pulmonalis* has a special tendency to hypertrophy.

An hypertrophic heart also presents an increased consistence of the muscular tissue. It feels rigid and hard as a board, and the greater consistence is also detected readily on cutting through the organ. The edges of the cut are widely separated. In many cases the muscle has a normal color; in others it is more of a reddish-brown, and under the microscope, the muscular fibres are then found to contain numerous yellow and brownish pigment granules, which are particularly abundant near the nuclei, and are often arranged in rows. Yellow, streaked patches are not infrequently observed macroscopically, and correspond to groups of fatty muscular fibres. These changes are secondary, but, nevertheless, very important, as they diminish the functional power of the heart.

The hypertrophy is generally most distinct in the left ventricle. On account of their slighter muscular development, the right ventricle and the auricles have a greater tendency to dilatation changes.

In *hypertrophy of the left ventricle*, the heart assumes a conical or cylindrical shape, and projects far into the left thorax. The apex is situated lower, and more to the outside than normally, and this is associated necessarily with a lower position of the diaphragm and the left lobe of the liver. Furthermore, the apex is formed exclusively of the left ventricle. A greater portion of the surface of the heart is in immediate contact with the wall of the thorax, because the anterior border of the left lung has been pushed to the outside. In considerable hypertrophy of the left ventricle, the lower lobe of the left lung may undergo so much pressure that it is deprived in great part of its atmospheric contents. The right ventricle is affixed to the left like a sort of insignificant appendix. Upon transverse section, it is readily seen that the anterior longitudinal sulcus no longer corresponds to the boundary between the right and left ventricles, but that the septum ventriculorum projects so strongly into the right ventricle that the latter appears narrowed in the highest degree.

In *hypertrophy of the right ventricle*, the left ventricle is pushed backwards, and upon opening the thorax it is almost exclusively the anterior surface of the right ventricle which comes into view. The shape of the heart changes; its angles are smoothed, and the shape becomes quadrilaterally rounded. The heart has grown particularly in width. The apex is lower than normal, and belongs chiefly to the hypertrophic ventricle.

*Hypertrophy of both ventricles* may lead to such an increase in the size of the heart that its anterior surface may be in contact with the wall of the thorax from the right mammary line to the left axillary line. The weight of the organ may even be increased fourfold, Hope having reported a case in which the heart weighed 1,250 gm., and Stokes one weighing 1,980 gm. (average normal weight 300 gm.). According to Rokitsansky, the greatest thicknesses hitherto observed were:

Left ventricle,	. . . . .	4	cm.
Right ventricle,	. . . . .	2	"

Left auricle, . . . . .	0.67 cm.
Right auricle, . . . . .	0.45 "

v. Buhl observed the following measurements in one case:

Greatest length, . . . . .	14 cm.
Greatest width, . . . . .	14.5 "

A generally hypertrophied heart is known as *cor bovinum* s. *taurinum*.

It must also be mentioned that a generally hypertrophied heart assumes a lower and more horizontal position, because it exercises, on account of its increased weight, greater tension on the origin of the large vessels. In addition, the apex is situated more to the outside—changes which are present to a less extent in hypertrophy of the left ventricle. Not infrequently, there is considerable dilatation and tortuosity of the coronary arteries.

Advanced hypertrophy of the heart is readily recognized. The determination of slight grades is more difficult because the weight of the normal heart varies to a certain extent.

It should be borne in mind that in the foetus the right and left ventricles are equal in thickness, and it is not until after birth that the thickness of the right ventricle becomes less than that of the left. Until the age of eight years, the left ventricle is relatively thicker than in adults, a feature which Gerhardt explains by the narrowing of the aorta at the opening of the ductus Botalli.

According to W. Mueller, the weight of the heart at the time of birth averages 20.69 gm. in boys, and 18.24 gm. in girls. Thoma furnishes the following table:

Until the end of the 1st year, . . . . .	37 gm.
2d to 5th years, . . . . .	50 to 70 "
6th to 10th years, . . . . .	70 to 115 "
11th to 15th years, . . . . .	130 to 205 "
16th to 20th years, . . . . .	218 to 254 "
21st to 30th years, . . . . .	260 to 294 "
31st to 50th years, . . . . .	297 to 308 "
50th to 65th years, . . . . .	308 to 332 "
65th to 85th years, . . . . .	332 to 303 "

It is seen that the weight of the heart increases particularly at the period of puberty, and diminishes beyond the age of 65 years, but W. Mueller draws attention to the fact that in advanced life the adipose covering of the heart increases. The heart is always lighter in the female than in the male at the same age, but, according to Mueller, the sexual difference only begins in the 6th year.

According to Bizot, the following are the dimensions of the heart from the 20th to the 60th years:

	IN MALES.	IN FEMALES.
Length of the heart, . . . . .	85 to 90 mm.	80 to 85 mm.
Width of the heart, . . . . .	92 to 105 "	85 to 92 "
Thickness of the heart, . . . . .	30 to 35 "	30 to 35 "
Thickness of the left ventricle at the base, . . . . .	10.1 "	9.8 "
Thickness of the left ventricle at the middle, . . . . .	11.6 "	10.8 "
Thickness of the left ventricle at the apex, . . . . .	8.4 "	7.9 "
Thickness of the right ventricle at the base, . . . . .	4.5 "	3.7 "
Thickness of the right ventricle at the middle, . . . . .	3.1 "	2.8 "



	IN MALES.		IN FEMALES.	
Thickness of the right ventricle at the apex, . . . . .	2.5	mm.	2.1	mm.
Thickness of the septum ventriculorum at the middle, . . . . .	11.0	"	9.0	"

In the casual determination of the dimensions of the heart, we may remember Laennec's statement that it corresponds approximately to the right fist of the individual.

II. ETIOLOGY.—Hypertrophy of the heart always develops when there is a continued increased call upon the activity of the organ. Under similar conditions the same change is found in the muscles of the body. The hypertrophy is partial or total according as the increased work is required of individual parts of the heart or of the entire organ.

In the majority of cases we have to deal with circulatory disturbances, but there are also cases in which the hypertrophy results from excessive acceleration of the movements of the heart. Thus cardiac hypertrophy develops gradually in hysterical and nervous individuals, who suffer from frequent attacks of palpitation. Continued mental excitement, excessive ingestion of coffee, tea, alcoholics, and immoderate use of tobacco may act in the same way.

Cardiac hypertrophy is observed in individuals who are addicted too strongly to the pleasures of the table. This was formerly regarded as the result of so-called plethora, inasmuch as the mass of blood was supposed to become abnormally large, and excessive work was entailed thereby upon the heart. This view does not appear to be correct, and it is explained more plausibly by the directly increased nutrition of the heart, as the result of high living.

Duroziez states that cardiac hypertrophy is a symptom of lead-poisoning, and this was corroborated by Roblot. But it is, to say the least, still uncertain whether the process must not be attributed to circulatory disturbances, inasmuch as contraction of the muscular coat of the vessels occurs, according to some writers.

The following are the circulatory disturbances which give rise to hypertrophy of the left ventricle:

*a. Diseases of the Aortic Valves.*—Hypertrophy of the left ventricle develops in aortic insufficiency, because the heart, at each systole, must propel a larger amount of blood into the aorta, *i. e.*, the normal amount in addition to that which has regurgitated into the left ventricle during the preceding diastole of the heart. We have previously stated that dilatation of the left ventricle is the first change which occurs in this valvular affection. In stenosis of the aortic orifice, the obstacle and the call for increased activity of the left ventricle are furnished directly by the stenosis.

*b. Stenosis of the Aorta.*—This occurs congenitally at the opening of the ductus Botalli, where it leads occasionally to occlusion of the aorta. Compression of the aorta by tumors has the same effect, as an abnormal obstruction must be overcome by the left ventricle.

*c. Abrupt dilatation of the aorta* or one of its main branches, as in aneurisms, diffuse dilatation of the aorta and larger branches, will also result in cardiac hypertrophy, because increased power is thereby required on the part of the left ventricle.

*d. Affections of the walls of the aorta and arteries,* when they lead to loss of elasticity of the walls, thus extinguishing a part of the propel-

ling force, and which must be assumed by the heart. This includes cardiac hypertrophy from arterio-sclerosis.

*e.* According to Larcher, who has been corroborated by many others, left ventricular hypertrophy develops as the result of pregnancy, but retrogresses after delivery. The introduction of the placental circulation has been regarded as the cause, but the pressure of the gravid uterus on the abdominal arteries should not be entirely overlooked. Other authors have denied the occurrence of the symptom in question. At all events, attention is merited by Gerhardt's statement that in pregnancy the diaphragm is abnormally high, so that the anterior surface of the heart is pressed against the chest-wall, and may give rise to apparent enlargement of cardiac dulness. Duroziez states that the hypertrophy continues after parturition so long as the mother nurses, and that women who have borne many children have a larger heart than those who have given birth to but one or two.

*f. Renal diseases.*—Hypertrophy of the left ventricle is observed most frequently in contracted kidneys, and this is explained, according to Traube, on the theory that a part of the renal capillaries are destroyed by the contracting and atrophic process, thus imposing more work upon the left ventricle. This seemed to agree with the observation that hypertrophy of the left ventricle is often observed in congenital absence of one kidney. Hahn, who has recently collected the histories of 37 cases, comes to the conclusion that, in the absence of one kidney, hypertrophy of the heart will be wanting only when the kidney which is present completely compensates for the absent one. Recently, however, opposition is being made to Traube's theory. v. Bamberger had shown previously that cardiac hypertrophy also occurs in chronic parenchymatous nephritis when atrophic processes in the kidneys are not present. Riegel has shown that hypertrophy develops very rapidly in acute nephritis, and this was corroborated by Friedlaender in autopsies upon acute scarlatinous nephritis, and by Leyden with regard to acute nephritis after typhoid fever. It would seem, therefore, as if, in consequence of renal disease, certain constituents are retained in the blood which irritate the heart muscle, and thus produce hypertrophy. The subject will be discussed more fully in the chapter on renal diseases. Cardiac hypertrophy has also been noticed in hydronephrosis and renal calculi.

The causes of hypertrophy of the right ventricle must be sought particularly in diseases of the orifice of the pulmonary artery, its trunk, and the pulmonary capillaries. These include stenosis of the pulmonary orifice, pulmonary insufficiency, stenosis of the pulmonary artery by tumors, aneurism and arterio-sclerosis of the pulmonary artery, in rare cases general dilatation of the branches of the pulmonary artery. Chronic diseases of the pleura and lungs also lead to hypertrophy of the right ventricle when the pulmonary capillaries are narrowed by compression, or have been destroyed after previous obliteration. Chronic exudations into the pleural cavities, emphysema, retraction of the lungs, bronchiectasis, spinal curvatures, etc., act in this manner. In phthisis, on the other hand, cardiac hypertrophy is rarely observed, because the heart is incapable of undergoing hypertrophy on account of the cachexia. Bäumler and Brudi have shown that extensive pleuritic adhesions lead not infrequently to cardiac hypertrophy, inasmuch as the power of locomotion of the lungs, and therefore their aspiratory action on the current of blood in the pulmonary artery, are interfered with. Under certain circumstances, the cause of right ventricular hypertrophy resides on

the farther side of the capillaries of the lung, as is sometimes observed in mitral lesions.

The cause of *auricular hypertrophy* is stenosis of the auriculo-ventricular orifices, which necessarily increases the resistance to the auricles.

*Hypertrophy of the entire heart* is observed when the causes of hypertrophy of the right and left ventricles are combined with one another. This occurs most frequently in simultaneous affection of the aortic and mitral valves.

But every cause which acts on a part of the heart may also lead to total cardiac hypertrophy, when the heart muscle fails and symptoms of stasis (or disturbance of compensation) develop. It will here suffice to show the necessary changes in two concrete examples.

If the muscular power of the hypertrophied left ventricle fails in aortic stenosis, some blood will remain in the left ventricle after each systole, thus furnishing the conditions necessary to stasis; or, in other words, to the elevation of the blood pressure in the left ventricle, and through this in the left auricle. Thence the elevation of blood pressure is propagated into the pulmonary veins, the capillaries of the lung, the pulmonary artery, and finally, the right ventricle, and thus furnishes a cause for (dilatation and) hypertrophy of the latter.

If, in mitral insufficiency, the power of the right ventricle no longer suffices to overcome the resistance furnished by the valvular lesion, some blood remains in the right ventricle after each systole, and thus stasis and elevation of blood pressure occur in the right ventricle, right auricle, and venæ cavæ, followed by œdema of the subcutaneous connective tissue, etc. It is not difficult to understand that this increases the resistance to be overcome by the left ventricle, and furnishes the conditions for its (dilatation and) hypertrophy.

Accordingly, if total hypertrophy is added to partial hypertrophy as the result of circulatory disturbances, we have to deal, in almost every case, with conditions of insufficient heart's action and disturbed compensation. But we must not conceal the fact that total hypertrophy is sometimes observed without preceding symptoms of stasis. This has been explained on the theory that, on account of the intimate connection between the muscular fibres of both ventricles, hypertrophic changes readily extend from one to the other.

Total cardiac hypertrophy is also observed in affections of the heart muscle itself. It is, therefore, found not infrequently in pericarditis and myocarditis. It is also met with occasionally in obliteration of the pericardium.

Finally, there are cases of mechanical hypertrophy in which no cause is revealed on autopsy. It seems almost as if we have to deal with a primary affection, so that the term primary or idiopathic hypertrophy has been used. But we usually learn from the history that the patients have endured excesses and great bodily labor, so that the apparently primary hypertrophy is really the result of circulatory disturbances, inasmuch as the capillaries are narrowed by the excessive muscular exertion, and the pressure in the domain of the aorta is thereby increased. Such observations have been made particularly in mountaineers, miners, blacksmiths, porters, sailors, and workers in vineyards. Cardiac hypertrophy in soldiers, as the result of excessive drilling and marching, has also been described. In certain regions the disease seems to be especially frequent, and this is probably the result, in part, of the occupation of the inhabi-



tants. Thurn found it frequently among English recruits, while Fraentzel, who described several cases as the result of forced marching during the Franco-Prussian war, could not corroborate Thurn's experience among German soldiers in times of peace. That such cases are not rare in North Germany is evident from the observations of Curschmann and Leyden and Zenker. I have repeatedly treated such patients in East Prussia, in Berlin, Jena, and Goettingen, and have been present at a number of autopsies. The left ventricle is affected most frequently, more rarely both ventricles, most rarely the right ventricle alone.

Psychical excitement (sorrow, care), excessive use of tobacco and alcohol, excessive venery, and gout are also mentioned as causes of idiopathic hypertrophy of the heart. According to isolated statements, cardiac hypertrophy is also hereditary, but this view is not well substantiated.

III. SYMPTOMS.—The symptoms of the primary disease and those of the cardiac hypertrophy must be kept strictly apart. We will here consider only the latter.

The subjective symptoms are unimportant, as the diagnosis is only possible from the objective symptoms.

In hypertrophy of the left ventricle, the cardiac region often projects more prominently, particularly in children and women whose ribs and cartilages are yielding. The apex beat is abnormally low, being situated often in the seventh or eighth intercostal space. At the same time it is situated more to the outside, so that it passes beyond the left mammary line and may extend to the left axillary line. The apex beat is also broader than normal (2.5 cm.), and the tips of two fingers no longer suffice to cover it. But while these changes in the apex beat belong rather to the accompanying dilatation of the left ventricle, it is also unusually lifting and resistant—a peculiarity characteristic of hypertrophy.

The pulsating elevations are usually not confined to the region of the apex beat, but the entire cardiac region, indeed the larger part of the left thorax undergoes a diffuse concussion at each systole. Not infrequently it can be recognized through the clothes, so that occasionally the entire body is shaken in systole, or a patient lying abed, produces systolic movement of the bed.

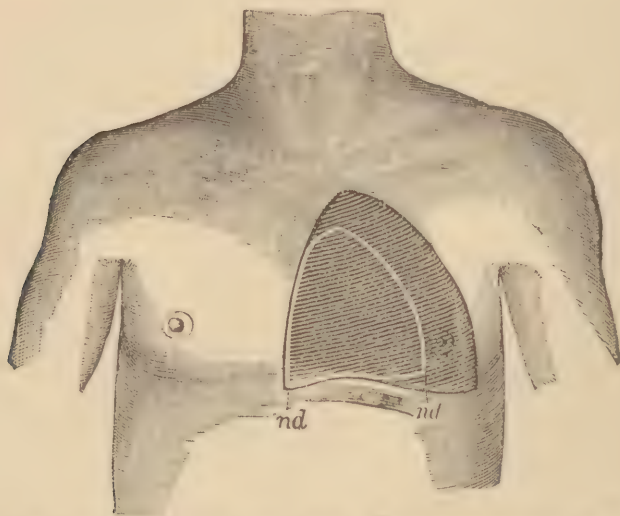
The movements of the heart can often be followed over a number of intercostal spaces, and, in addition, systolic retractions are observed not infrequently immediately adjacent to the left edge of the sternum.

In the lateral cervical region, there is usually vigorous throbbing of the carotids, caused by the abnormally high pressure in these arteries, which, in addition, often contain an unusual amount of blood. This also explains the fact that pulsation is visible in the smaller arteries, for example, the temporal, in which it is not found in healthy individuals, unless the heart's action is unusually vigorous.

On palpation, special attention should be paid to the changes mentioned in the breadth and strength of the apex beat. At times a short beat is felt in the second right intercostal space, alternating with the apex beat and therefore diastolic in character. It corresponds to an unusual development of the aortic semilunar valves. Over the carotids we often feel systolic thrill, which is also present when palpation is exercised gently and carefully. On auscultation a systolic murmur is heard in this situation. The examination of the pulse is very important; it is extraordinarily hard and tense.

Percussion shows enlargement of cardiac dulness in a direction from above downwards and to the left externally. The greater (relative) area of cardiac dulness often begins above at the second left costal cartilage, and extends downwards to the eighth or ninth rib. Externally to the left it may be sometimes followed as far as the left middle axillary line, but it always stops at the region of the apex beat. The small (absolute) cardiac dulness is also increased in proportion. The area of dulness has the shape of an elongated oval (Fig. 7). Important changes are observed on auscultation. The diastolic or second aortic sound is unusually loud: it is usually short, beginning and ending quickly, clear, and valvular. The increased action of the hypertrophic left ventricle corresponds, during diastole, to increased tension of the semilunar valves of the aorta, which is recognized on palpation as a diastolic beat; on auscultation, as intensification of the second aortic sound. The systolic ventricular sound is also changed not infrequently. It is often accompanied by a peculiar

FIG. 7.



Shape of the large (relative) area of cardiac dulness in hypertrophy of the left ventricle. The white line *nd* indicates normal cardiac dulness.

rattling sound (*cliquetis metallique*). This results from systolic concussion and vibration of the chest walls, and is found particularly in individuals with a yielding thorax which is capable of vibration, while it is absent when the ribs are broad, ossified, and immobile. The first ventricular sound is heard occasionally at some distance from the patient. The vigorous concussions of the chest walls are perceived actively during auscultation, inasmuch as the stethoscope and head are raised with each systole and fall back during diastole. In view of the great intensity of the heart sounds, it is not astonishing that they are propagated far beyond the cardiac region, so that they may be heard over the entire posterior surface of the thorax. But we must be careful not to regard this as pathognomonic.

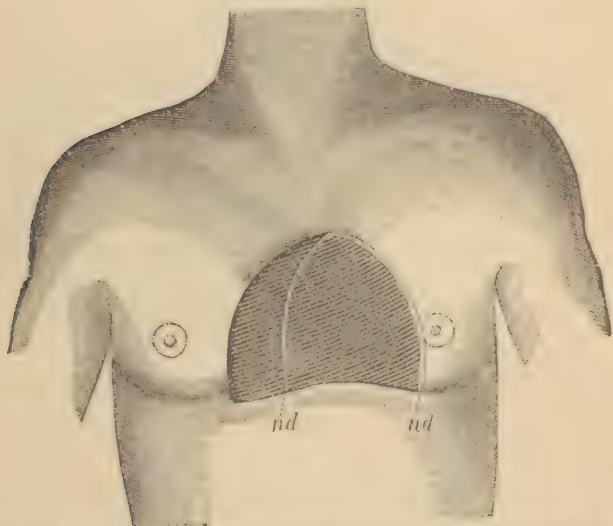
Instead of the systolic tone we occasionally hear over the carotid a systolic murmur which is explained by the excessive tension of the wall

of the vessel and its consequent irregular vibrations. The intensified diastolic aortic sound is usually propagated into the carotid. Over the small arteries we often hear a short systolic note, when the stethoscope is placed upon them without exercising any pressure. The systolic tone in the arteries may even be heard over the palmar arch.

Subjective symptoms are often entirely absent, except that palpitation and dyspnoea develop during violent exertion. Some patients complain of a feeling of constant tension and pressure in the cardiac region, which is intensified occasionally into a slightly painful sensation. Attacks of palpitation occur not infrequently without any special exciting cause, and may be accompanied by violent pain in the region of the heart, radiating sometimes into the left arm. Many patients are compelled to assume a certain position in bed, as the symptoms are usually especially severe in left lateral decubitus.

A rush of blood to the head often occurs. The patients complain of

FIG. 8.



Shape of the large (relative) area of cardiac dulness in hypertrophy of the right ventricle. The white line *nd* indicates normal cardiac dulness.

dizziness, dulness, spots before the eyes, ringing in the ears, occasionally of subjective pulsatile auditory sensations. There is often a tendency to hemorrhages, shown by repeated and profuse epistaxis and in women by profuse menstruation. Cerebral hemorrhage occurs comparatively often, but is usually preceded by changes in the cerebral vessels.\* The latter are also explained by the abnormally high blood pressure and the partly changed nutritive conditions.

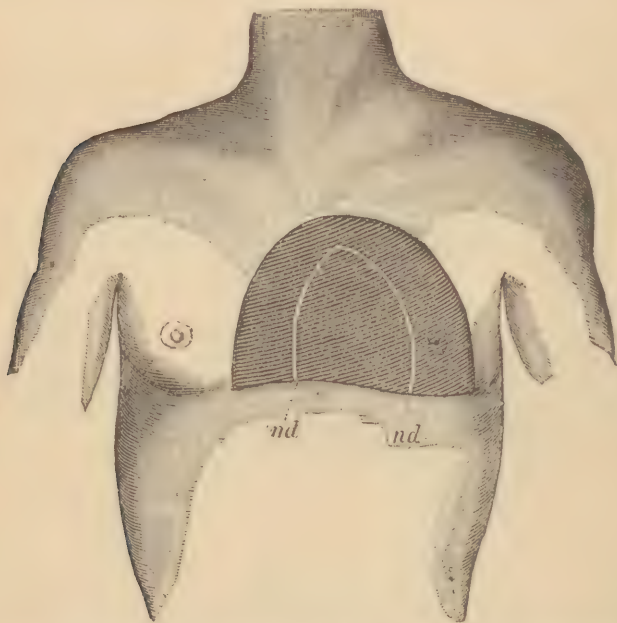
A suspicion of hypertrophy of the right ventricle is often roused on inspection from the fact that the diffuse apex beat extends very far to the right, so that it may be followed with the eye over the lower part of the sternum and even the adjacent parts of the right thorax. On palpation we feel not infrequently an abnormally active concussion of the parts of the thorax mentioned. In many cases there is also a short dias-



toxic beat confined to the second left intercostal space and attributed to excessive tension of the pulmonary valves. On auscultation, this is recognized by the intensification of the diastolic pulmonary sound. On percussion we notice extension of the heart to the right, the great (relative) cardiac dulness extending externally beyond the right edge of the sternum, and the cardiac resistance extending, at the level of the fourth costal cartilage, more than two cm. beyond the right edge of the sternum. The area of dulness of the heart has increased particularly in width, but very little in length (Fig. 8).

In right ventricular hypertrophy, the patients usually suffer from cyanosis and shortness of breath, because the pulmonary circulation is always affected. They also have a marked tendency to bronchial catarrh, and hemorrhages from the air passages are not infrequent.

FIG. 9.



Shape of the large (relative) area of cardiac dulness in hypertrophy of both ventricles. The white line *nd* indicates normal cardiac dulness.

The symptoms of hypertrophy of the entire heart consist, in great part, of a combination of the symptoms just described. The area of dulness may assume such dimensions that it extends from the right mammary line to the left axillary line, and from the second costal cartilage to the eighth and ninth ribs. At the same time the triangular shape of the cardiac dulness becomes rounded, quadrilateral, and almost trapezoidal (Fig. 9). In some cases Seitz detected pericardial friction murmurs, which he attributes to the rubbing of the abnormally large heart muscle against the parietal layer of the pericardium.

Very distressing conditions develop when the heart muscle possesses insufficient power to overcome the abnormal resistance to circulation. This is often indicated by the fact that the dyspnoea and palpitation become

constant and the pulse is often accelerated and irregular. Despite our endeavors we are unable permanently to combat the obstructions. (Edema develops, diuresis diminishes, occasionally albuminuria occurs. The liver increases in size, and symptoms of ascites soon present themselves. The patients cough a good deal, and not infrequently expectorate blood. Death from suffocation results from hydrothorax, oedema of the lungs, or pneumonia. In other cases life ends with signs of cerebral congestion, the patient passing into a condition of somnolence attended with convulsions. Sometimes we are able to obviate the danger, but we can merely delay the fatal termination more or less, and increasing cardiac insufficiency soon puts an end to life.

IV. DIAGNOSIS.—The recognition of cardiac hypertrophy is usually unattended with difficulty. The symptoms with regard to hypertrophy of the left ventricle are: a particularly broad and lifting apex beat, the apex beat lower than normal and passing beyond the left mammary line, intensification of the second aortic sound, enlargement of cardiac dulness in the direction from above downwards and to the left externally, hard and tense radial pulse. In hypertrophy of the right ventricle we find: widening of the diffuse apex beat over the sternum and beyond it to the right, widening of the area of dulness transversely, the greater (relative) cardiac dulness passing beyond the right edge of the sternum, intensification of the second pulmonary sound.

The mistakes which may be made with regard to cardiac dulness have been discussed under the head of pericarditis, and will be considered here very briefly.

Enlargement of cardiac dulness remains absent if the anterior borders of the lungs are fixed by adhesions and cannot be pushed to the outside by the enlarged heart. But this is only the case so far as regards the smaller (absolute) cardiac dulness, *i. e.*, that portion of the heart which lies immediately adjacent to the thorax, while the greater cardiac dulness is very little affected thereby. Increase of cardiac dulness may also be absent in pulmonary emphysema on account of the great distention of the lungs, and after due consideration of the etiology we must often be satisfied with making a probable diagnosis.

In other cases there may appear to be increased cardiac dulness on account of disease of adjacent organs, although the heart is normal in size. This may occur in infiltration of the borders of the lungs, encapsulated pleurisy, aneurisms of the aorta and pulmonary artery, and mediastinal tumors. Tumors situated behind the heart and pressing it forwards, may also apparently increase the area of dulness. In such cases we must consider the other symptoms, the usually more irregular shape of the area of dulness, and must not undervalue the diagnostic importance of the etiology.

In total hypertrophy of the heart, it may be doubtful whether the increased area of dulness is owing to pericarditis or cardiac hypertrophy. We must then consider the development of the affection, the characteristic shape of the area of dulness in pericarditis and the relation of the apex beat to the dulness. Friction murmurs favor the diagnosis of pericarditis, as the rubbing murmurs caused by the heart muscle, which have been described by Seitz, must be very rare. Increase of the area of dulness in the upright position points towards pericarditis, although it has been observed in a few cases of extensive hypertrophy.

V. PROGNOSIS.—Cardiac hypertrophy must be regarded as a favorable sign in so far as it allows the possibility of overcoming the abnormal

resistance to the circulation. Unfortunately, the prognosis does not continue favorable. In almost every case a stage develops in which the heart's action fails, and though such conditions may be relieved temporarily, the force of the heart cannot be maintained permanently.

VI. TREATMENT. As cardiac hypertrophy is an attempt on the part of nature to relieve existing circulatory disturbances, it would be wrong to attempt to prevent its development. On the contrary, we should endeavor to maintain the hypertrophy and the vigor of the heart unchanged, not indeed by medicinal agents, but by a rational regimen. Excessive labor should not be imposed upon the heart, and strict bodily and mental rest should be recommended. Hard work, mountaineering, exhausting walks, dancing, riding, must be interdicted. Care should also be exercised in taking cold baths, as some patients are seized in the bath with sudden attacks of oppression and palpitation, leading to the danger of drowning. Cold frictions, morning and evening, are indicated for many patients.

We should prohibit the use of stimulants, particularly coffee and tea, nor is smoking allowable. The articles of diet should be such as are readily digested and not very saculent. We may recommend milk, eggs, soup, lean meats, and cooked fruit to aid the action of the bowels, while farinaceous foods, vegetables, and fatty articles should be avoided. Heavy meals should be avoided; it is better to eat oftener, but little at a time. In well-fed individuals, addicted to the pleasures of the table, we may recommend whey and grape cures (for example, in Merau, Montreux, Vevey, Bex, Duerkheim, etc.). A sojourn in the mountains acts remarkably well in some cases.

Mild laxatives should be given in constipation, since otherwise a rush of blood to the head is apt to occur.

In attacks of palpitation, the patients must be kept absolutely quiet, and an ice bag applied over the heart. Some patients experience considerable relief from constantly wearing a flask filled with cold water and fitted to the shape of the chest, but the water should be frequently renewed. Little can be expected from the use of setons, issues, and other derivatives in the cardiac region. Digitalis in large doses should be administered if the palpitation continues.

We must especially make use of digitalis (its effects being always carefully watched) when the heart's action becomes insufficient, and its effects far surpass those of the other remedies, such as caffeine, adonis vernalis, convallaria majalis, quinine, veratrine, arsenic, antimony, delphinium, etc. Diuretics, diaphoretics, and drastics are indicated if edema and other symptoms of stasis develop.

### 3. *Atrophy of the Heart.*

I. ETIOLOGY.—Atrophy of the heart is a diminution in size and partial disappearance of the heart muscle. It affects usually the entire heart, partial atrophy being rarer. The latter form is found in the left ventricle in mitral stenosis, evidently because the ventricle gradually accommodates itself to the smaller quantity of blood which flows into it during diastole.

Like the majority of other organs, the heart undergoes senile atrophy in advanced age. It is also observed when the organism passes into a marantic and cachectic condition as after carcinosis, tuberculosis, long continued suppuration, severe typhoid fever, dysentery, diabetes, losses



of blood, stenosis of the œsophagus and similar affections, for example, refusal of food. Occasionally a sort of compression atrophy develops. This is observed in mediastinal tumors, long standing pericarditic exudations, callous thickenings of the epicardium, and excessive development of sub-epicardial fat. Stenosis of the coronary arteries may also lead to atrophy of the heart on account of the insufficient nutritive supply.

Rokitansky first called attention to the fact that abnormal smallness of the heart may be congenital. He observed it particularly in delicate, amenorrhœic women, associated with defective development of the genital system. But Virchow was the first to lay stress upon the fact that imperfect development of the heart and arteries is intimately connected with chlorosis. I have also made several autopsies upon pale men in whom the dimensions of the heart were approximately those of a child of five or six years. As we have to deal in such cases with a congenital condition and not with a retrogressive change, it should not, strictly speaking, be termed atrophy, and Virchow therefore applied the term hypoplasia. Virchow also found hypoplasia of the heart in bleeders (those suffering from hæmophilia).

Brehmer maintains that congenital atrophy of the heart predisposes to pulmonary phthisis, but this is disputed by v. Mayer, who holds that cause and effect have been mistaken for one another.

II. ANATOMY.—An atrophic heart is small in size, has thin walls and diminished weight. According to Wanderlich, the heart is atrophic if it does not reach 200 gm. in weight (in the adult). In phthisis, Engel found the weight of the heart reduced to a fourth of the normal, the diminution affecting chiefly the left ventricle.

The pericardium is often found filled with a clear fluid. This was formerly regarded as an hydro-pericardium *ex vacuo*, but in our opinion the transudation is the result of the primary disease.

The surface of the heart is often folded and wrinkled, particularly in those places in which the epicardium is thickened into milk spots; the heart has been compared to a dried pear. As a rule, the sub-epicardial fat has disappeared, occasionally it forms an atrophic mucoid or gelatinoid mass. Increase of the sub-epicardial fat is observed much more rarely. The coronary arteries are often remarkably sinuous.

The heart muscle is often pale, but firm and rigid; its color sometimes resembles that of muscular tissue which has been macerated for some time in water. In other cases it has a rusty brown or dark ochre yellow color, particularly in old people, and those who have died of cancer or phthisis. This discoloration is caused by profuse yellow and brown pigment granules in the muscular fibres, partly arranged in rows above one another and accumulated especially at the ends of the nuclei of the fibres. They are regarded as the remains of the muscle coloring matter, but careful investigation concerning them is desirable. These changes have been termed brown atrophy, pigment atrophy, or pigment degeneration of the heart muscle.

Microscopical examination sometimes reveals other degenerative changes in the atrophic muscular fibres. Friedreich found, particularly in the cardiac atrophy of cancer and tuberculosis, that the muscular fibres had lost their transverse striae, and had become converted into homogeneous, colorless cylinders, the nuclei of which in part had disappeared. He applied to these changes the term sclerotic atrophy, because it was characterized macroscopically by striking firmness of the muscle, and upon transverse section by smoothness, waxy gloss, and its

almost transparent, grayish-red appearance. In other cases the muscular fibres are partly granular, partly fatty, or, as Virchow described in one case, have undergone waxy degeneration. At all events these secondary changes indicate that atrophy of the heart depends not alone on diminished size of the muscular fibres, but that it is caused partly by degenerative processes.

The aortic semilunar valves also present atrophic changes not infrequently, inasmuch as they are extremely thin and delicate, occasionally are even perforated and fenestrated. The free edge of the leaflets of the mitral and tricuspid valves are often found rolled inwards.

The cavities of the heart are generally smaller than normal, inasmuch as they accommodate themselves to the quantity of blood which, as a rule, is diminished. This has also been termed concentric atrophy of the heart. In eccentric cardiac atrophy, the lumen of the heart appears enlarged; in simple atrophy, the cavities of the heart are unchanged. Eccentric atrophy is rarer than simple atrophy; it occurs most frequently in old age, provided the amount of blood is not notably diminished.

III. SYMPTOMS; DIAGNOSIS; PROGNOSIS; TREATMENT.—A number of symptoms have been mentioned by means of which atrophy of the heart may be recognized, but these are in part purely theoretical, in part they are accidental complications. Laennec taught that syncopal attacks in hypochondriacal individuals depended on atrophy of the heart, and Hope made a similar statement with regard to nervous and hysterical individuals. When we read further that feeble, trembling, or absent apex beat, low heart sounds, attacks of palpitation, small pulse, etc., occur in cardiac atrophy—who would venture a diagnosis on such doubtful symptoms?

Among objective symptoms we would naturally expect diminution of the area of cardiac dullness, but every experienced observer who has the opportunity of controlling his diagnoses by autopsies, knows that little weight can be attached to this symptom, even when diminution of the area of dullness by pulmonary emphysema may be excluded. The diagnosis, therefore, depends solely on experience, and cannot possess more than a certain degree of probability. The prognosis is unfavorable on account of the grave primary affections. If treatment must be considered at all, the indications are to strengthen the action of the heart and secure complete bodily and mental rest.

#### 4. *Fatty Heart, Cor Adiposum.*

(*Adipositas s. Lipomatosis s. Obesitas cordis. Lipoma Capsulæ cordis. Virchow. Atrophia cordis lipomatosa, Orth.*)

I. ANATOMICAL CHANGES.—On the healthy heart we always find a moderate amount of fat in the subepicardial connective tissue, particularly in the grooves of the heart and in the vicinity of its chief vessels at the lower edge of the right ventricle and near the apex. Excessive increase of this fat leads to the development of fatty heart. The fat then increases considerably, not alone in the situations mentioned, but also extends over the surface of the ventricles. The right ventricle is first surrounded by a massive layer of fat, later the left ventricle is similarly involved. Finally, the entire heart is surrounded by a thick capsule of fat, which may attain a diameter of more than one cm. Not infrequently the fat has an intense sulphur-yellow, almost icteric color, while in ether

cases it is pale-yellow. Upon section through the heart muscle we often find that the proliferation of fat is not confined to the subepicardial connective tissue, but has also penetrated along the intermuscular fibrous septa in the depth of the heart muscle. In many cases, also, the muscular tissue of the heart is extremely diminished, and has a brownish-yellow, dull color. The heart muscle is reduced occasionally to a very thin stripe, so that it is astonishing that it could effect the circulation of the blood. We can also readily understand that such a muscle is especially predisposed to rupture. Endarteritic changes in the coronary arteries and aorta are noticed not infrequently.

On microscopical examination we often find appearances which indicate atrophy, fatty degeneration, and, finally, disappearance of the muscular fibres, as the result of being surrounded and compressed by the adipose tissue. It is evident, therefore, that an originally fatty heart may be associated with fatty degeneration of the muscular fibres. In one case Kennedy found the right pneumogastric healthy, the left one extremely fatty.

The other organs present very different and often almost antagonistic conditions. While some bodies present the appearances of excessive emaciation, in others we are struck with the marked obesity of various organs, for example, the mediastinum, omentum, mesentery, liver, subcutaneous connective tissue, etc. According to Smith's observations, drops of free fat are sometimes found in the blood in such cases, but on a perusal of Stokes' cases we cannot avoid the suspicion that we have to deal with accidental uncleanness at the autopsy. Furthermore, recent statements of a similar tenor do not withstand unbiassed criticism.

II. ETIOLOGY.—Among the causes of this affection, general obesity must be taken into consideration in the majority of cases (vide Vol. IV.).

It may here be mentioned that it is found particularly in those who are very much addicted to the pleasures of the table and, at the same time, have very little bodily exercise, or in those who adopt an improper diet, particularly one rich in hydrocarbons (farinaceous articles, sweets, etc.). It is also well known that drinkers of beer, brandy, or wine have a tendency to obesity.

Among other, but much less important, causes we may mention losses of blood and exhausting discharges, and anaemic and cachectic conditions (chlorosis, phthisis, carcinosis, serofula, Addison's disease, etc.). In this class of cases the heart is occasionally found fatty, while signs of emaciation are manifest in the subcutaneous cellular tissue and muscles.

Obesity and fatty heart also develop in women suffering from amenorrhoea and sterility, or in those who have recovered from a child-birth, or have reached the menopause.

Due weight must also be attached to hereditary predisposition. Daily experience teaches that one individual will gain in weight under certain conditions, while another will present a lean appearance.

Fatty heart occurs more frequently in men than in women. As a rule, the individuals have passed the age of forty years. Blachez reports the case of a girl, *æt.* 16 years, who suffered from fatty heart as the result of drink, and died in consequence.

III. SYMPTOMS.—In not a few cases the disease has no special symptoms, and is found accidentally at the autopsy. In others, there are no previous striking symptoms, but the disease is the cause of sudden death; a relatively large number of cases of so-called spontaneous rupture of the heart depend upon fatty heart, and the consequent diminished power of resistance of the heart muscle. In a third group of cases the most distressing conditions develop on account of the temporary or



permanent inability of the organ to fulfil its function. The symptoms of insufficiency of the heart's action develop. As a matter of course, these are in no wise characteristic of fatty heart, and may be expected so much the more, the greater the amount of muscular substance which has disappeared on account of proliferation of fat, the larger the amount of fat, and the more it interferes with the heart's action. Sometimes the symptoms develop suddenly after some violent exertion, for which the heart's action has proved unequal. At times the heart recovers temporarily; at other times its power constantly diminishes more and more.

In certain cases an excessive panniculus adiposus indicates that the circulatory disturbances present are dependent on fatty heart. In other cases the history must be taken into consideration, special attention being paid to the question of alcoholism. Not without reason some of the older English physicians called attention to the pale complexion, particularly to the pale-yellow color of the face. The latter is said to be especially distinct upon the cheeks, immediately beneath the eyes and near the naso-labial fold. Great importance has also been attached to the presence of the arcus senilis, which appears as a light coloration at the edge of the cornea, and is caused by fatty degeneration of the corneal corpuscles and the intercellular substance. But this change occurs with such uniformity in old people, that in them no diagnostic significance can be attached to it, but in young individuals it is valuable in so far as it discloses a tendency of the organism to fatty changes.

Insufficiency of the heart's action is also shown by the small amount of blood in the arteries and the abnormal distention of the veins.

We also notice feebleness or absence of the apex beat, and a diffuse impulse of the heart. Percussion shows not infrequently an enlargement of cardiac dulness, because the flabby muscle has a tendency to dilatation of the cavities. The heart sounds generally have but little intensity, and the first ventricular sound is not infrequently converted into a systolic murmur. Some authors attribute this to fatty changes in the papillary muscles, which give rise to insufficiency of the venous valves. In our opinion, however, it results from arrhythmical vibrations during systolic contraction of the heart muscle.

The cervical veins are distended, and may present undulatory movements. The radial pulse is abnormally small and feeble. Attacks of palpitation of the heart occur very often, sometimes spontaneously, sometimes after trifling bodily or mental exertion. They may be associated with pain around the heart, radiating not infrequently towards the periphery, and then almost always into the left arm. In certain cases, profuse sweats and marked fall of bodily temperature are observed. In these conditions dyspnoea is usually prominent, and it may closely simulate the history of an asthmatic attack (*asthma cardiale* of some authors).

English writers have called attention to the diagnostic importance of three symptoms, viz.: slow pulse, pseudo-apoplectic attacks, and Cheyne-Stokes breathing, but these appear to be rarer on the Continent than in England.

The retardation of the pulse may be extreme. In Cornil's case the pulse fell to fourteen beats a minute for several days. A frequency of eight a minute is reported in some of the older cases. This diminution may be permanent, or occur only at times, usually in association with the syncopal and pseudo-apoplectic attacks. In Cornil's case there were attacks of unconsciousness, respiratory disturbances, and convulsions, which lasted as long as twenty seconds, and during which the heart is said to have stopped beating entirely. I have treated individuals who predicted the occurrence of cerebral attacks hours before by the appearance

of striking retardation of the pulse. v. Dusch found that the retardation of the pulse continues after the onset of febrile diseases. Irritation of the vaso-motor nerve centre must be regarded as the cause of the abnormal slowness of the pulse. It is produced by the cerebral anæmia which, as has been proven experimentally, results in slowed movement of the heart.

During the pseudo-apoplectic attacks the patients, in many cases, suddenly lose consciousness and fall. If they recover, complete consciousness is usually restored with striking rapidity. Paresis of certain limbs is sometimes left over for a little while; more rarely permanent hemiplegia follows, and must be attributed to encephalorrhagia. This may occur so much the more readily because fatty degeneration of the smaller cerebral vessels is observed very often even in young persons, who are suffering from fatty heart.

The number and duration of the pseudo-apoplectic attacks vary greatly. In many cases weeks and months elapse before they are repeated, in others they occur several times in the course of a day. In one of my patients ten to fifteen attacks occurred in one day. The patient often stopped in the middle of a sentence, let the head fall (the eyes being closed), and the extremities also fell powerless. Upon coming to, he continued his sentence and had no knowledge of the seizure. The more rapidly the attacks follow one another the shorter is their duration, and there are numerous gradations from rapidly passing unconsciousness to attacks which last many hours. Sometimes the patient feels the approach of the attack. We have previously mentioned that slowness of the pulse indicates the approaching seizure. There are also peculiar premonitory sensations, and the patients may learn to prevent the attack. In Stokes' well known case, as soon as the patient felt the onset of the attack, he assumed the knee-elbow position, dropping the head far downwards; this generally warded off the attack.

Twitchings in the limbs appear occasionally during the attack; there are almost always changes in the pulse and disturbances of respiration. The Cheyne-Stokes' respiration is readily recognized. It consists in the regular alternation of phases and pauses of respiration. The former begin with superficial respirations; then they become deeper and deeper, assume a dyspnoeal character, often become sighing and stertorous, and gradually subside into apnoea (cessation of respiration). Leube first noticed that the pupils are contracted during the apnoea, and Traube observed that twitchings may occur in individual groups of muscles towards the close of the pause in respiration. According to Fraentzel the apnoea may last as long as forty seconds. Cheyne-Stokes' breathing sometimes appears only during sleep, in other cases sleep or disturbance of consciousness occurs during the pauses in respiration. Fraentzel first noticed that the phenomenon could sometimes be produced by the use of narcotics. I have been in the unpleasant position of noting the correctness of this statement in the case of one of my patients. Cheyne-Stokes' breathing is also observed not infrequently during the pseudo-apoplectic attacks. Sometimes the phenomenon is only temporary, but it may also last weeks and even months.

Traube was the first to explain this symptom by anæmia of the medulla oblongata, the centre of the respiratory and circulatory nerves. But as the phenomenon may also occur in other conditions, it is evident that it is not pathognomonic of fatty heart. Among others it is found in compressing diseases of the contents of the skull; for example, acute hydrocephalus, cerebral tumors, hemorrhage, meningitis, and in toxic affections (uræmia, cholæmia).

Anæmia of the central nervous system in fatty heart is sometimes not confined to the brain, but appears to affect the spinal cord.

The cardiac affection may last for years, and every experienced physician meets with cases in which improvement and exacerbation alternate for several years.

The fatal termination is very often sudden. Among Quain's collected cases, sudden death occurred fifty-four times, or a little more than sixty-six per cent. Death may occur from rupture of the heart, as was observed by Franz during childbirth. In other cases death is the result of cerebral hemorrhage. Pseudo-apoplectic attacks may also be the direct cause of death.

In some cases there is repeated and profuse epistaxis, which favors the fatty changes and the impairment of strength, and accelerates the

fatal termination. A chronic and very distressing course develops when signs of insufficient heart's action appear more and more prominently, and lead to death with symptoms of stasis: oedema of the skin and serous cavities, enlargement of the liver, diminished diuresis, albuminuria, bronchial catarrh, hemorrhagic infarctions, pneumonia, pulmonary oedema, somnolence, convulsions, etc.

IV. DIAGNOSIS.—As a rule, the diagnosis can only be made with a certain degree of probability. Cases in which striking retardation of the pulse, pseudo-apoplectic attacks, and Cheyne-Stokes' breathing occur in combination are not very frequent, and, as a matter of course, very little can be inferred from a single one of these symptoms. If the signs of insufficient heart's action develop, we must, first of all, exclude myocarditic changes, and it will then be of special value to take into consideration the clinical history and etiology, and the signs of general obesity.

V. PROGNOSIS.—The prognosis is usually unfavorable, for, as a rule, the patients are seen in such a late stage of the disease, that radical treatment is neither advisable nor does it promise permanent benefit. In addition, many patients will not give up their bad habits.

VI. TREATMENT.—There is generally no opportunity for prophylactic measures, as the patients do not seek advice until after they have grown obese. However, the prophylactic measures and the treatment of obesity and fatty heart coincide with one another. For details we refer to Vol. IV., article Obesity. It may here be mentioned that special importance must be attached to a restriction of the amount of nutriment and the greatest possible exclusion of hydrocarbons, while fats may be ingested ad libitum. In addition, moderate exercise, but not excessive, particularly in the beginning, when the heart muscle must still be spared as much as possible. The waters of Marienbad, Kissingen, Homburg, Wiesbaden, Tarasp, and Karlsbad have a good reputation in this affection.

If insufficiency of the heart's action develops, we should order digitalis (as infusion 2 : 200, one tablespoonful every two hours) alternating with *cafeinum citrico-benzoicum* (glycerin., aq. destill., āā 5.0, one syringeful morning and evening), and with *adonis vernalis* (inf. 5 : 150, one tablespoonful every two hours). When symptoms of stasis are prominent, we should resort to diuretics (*Acet. Scillae*, 30.0. *Kali carbonic.*, q. s. ad perf. saturationem, adde *Aq. Petroselin.*, 150.0. *Oxymel Scillitic.*, 20.0. M. D. S. One tablespoonful every two hours), to drastics (inf. *sennae comp.*, 180.0. *Natri sulph.*, 20.0, M. D. S., one tablespoonful t. i. d.), and to diaphoretics.

When symptoms of cerebral anæmia appear, the patient's head should be kept low and stimulants given internally, for example, strong wines in teaspoonful doses, sulphuric ether, five drops on sugar repeated: tinct. *valerian. æth.*, twenty drop sin one drachm sugar-water; camphor, 1.0. *Ol. amygdal.*, 10.0. M. D. S., one syringeful subcutaneously; ammonia or eau de cologne by inhalation, rubbing the temples with eau de cologne, hand baths and foot baths of mustard (50.–100. of coarsely ground mustard for each bath), mustard poultices to the calves, the epigastrium, etc.

Asthmatic attacks often yield rapidly if the patients lie down in a dark room. We may also apply an ice-bag to the præcordial region, give pieces of ice, also ices internally by the teaspoonful, perhaps allow a cup of very strong coffee. Great care should be exercised in the use



of morphine and narcotics, as they readily induce Cheyne-Stokes respiration and cerebral anæmia.

If the threatening symptoms have been relieved, it is often very advisable to make a prolonged use of small doses of digitalis, for example, fol. digit. pulv., 2.0. Ferri lactie., Kali nitric., aa 10.0. Ext. rhei comp., 0.5. Pulv. althææ, q. s. ut f. pil. No. 100. D. S., two pills to be taken t. i. d.

### 5. *Inflammation of the Heart Muscle. Myocarditis.* (*Carditis Musculosa*.)

I. ANATOMICAL CHANGES.—According to the extent of the inflammatory process, we distinguish diffuse and partial myocarditis; according to its course, acute and chronic myocarditis; and according to the situation of the inflammation, we have to deal with a parenchymatous inflammation, *i. e.*, one which occurs in the muscular fibres, and with an interstitial inflammation, which is situated in the intermuscular connective tissue. Not infrequently, however, there are transitions between these various forms.

The changes in the heart muscle, described by Virchow as cloudy swelling, are generally included in the category of the diffuse form of acute parenchymatous myocarditis. The heart muscle appears somewhat swollen, often has a dull, shining, or lardaceous fatty appearance, and is not infrequently extremely brittle. On microscopical examination the individual muscular fibres are found to be swollen and to have lost their transverse striation, and are filled with innumerable, densely aggregated granules. On the addition of acetic acid or dilute potash, the granules disappear and are converted into a homogeneous, transparent mass, thus proving their albuminoid character. There may also be a slight increase of the nuclei of the fibres.

If the primary affection is especially severe and prolonged, the changes do not remain in the stage of cloudy swelling. The individual granules are converted into little drops of fat, no longer dissolve in acetic acid and potash, and are stained gray or black by osmic acid. The nuclei take part in the fatty degeneration, and if the latter process is severe enough, the muscular fibre constitutes a cylinder in which nuclei are no longer demonstrable and which is densely filled with larger drops of fat.

Acute diffuse parenchymatous myocarditis occurs most frequently in febrile infectious diseases, apparently as the result of the infection and not of the accompanying fever. It would not be in accordance with the facts, however, if we assume the existence of this change in all infectious diseases. We have previously stated that the intensity and duration of the disease exert an influence, but there is also evidently a third important factor, *viz.*: the individual power of resistance of the heart muscle. It is unnecessary to say that the changes in question are not unimportant. They will necessarily enfeeble the power of the heart, and may lead to its complete paralysis. In many infectious diseases this constitutes the chief danger. At present, however, we will not discuss this form any further.

Diffuse acute interstitial myocarditis, also, is almost always associated with infectious diseases. Leyden showed recently that, in diphtheria, there is a proliferation of round cells in the intermuscular connective tissue of the heart, which may even give rise, in places, to disappearance of the muscular fibres. Goodhart describes the case of a child, *æt.* 3½ years, who died of scarlatinal nephritis, and in whom the interstitial connective tissue of the left ventricle was diffusely infiltrated with pus. Probably many other infectious diseases may give rise to similar changes. These conditions cannot be diagnosed with certainty during life, as the signs of acute dilatation of the heart, heart failure (soft heart sounds, feeble apex beat, small pulse) are as doubtful in their significance as reduplication of the first sound, and the so-called rhythm *du galop*.

Acute circumscribed myocarditis is usually purulent (myocarditis purulenta, abscess of the heart), and, in the majority of cases, the result of embolic processes in the branches of the coronary arteries. It follows, therefore, that it is an inter-

stitial myocarditis, though the muscular fibres in the vicinity of the abscess are not free from inflammatory changes. This embolic or metastatic myocarditis is observed with the greatest relative frequency in ulcerative endocarditis, in which colonies of bacteria separate from the site of inflammation on the endocardium, and are carried into the coronary arteries. This also occurs in pyæmia, puerperal fever, splenic fever, glanders, acute articular rheumatism, diphtheria, typhoid fever, and in purulent and gangrenous processes in the air passages.

Sometimes cardiac abscesses, for which no cause can be ascertained, attain very considerable dimensions.

The embolic abscesses of the heart, in ulcerative endocarditis, are multiple, and not infrequently present in astonishing numbers. Their appearance varies according to their age. The most recent are very gray, or grayish-yellow dots or stripes, which consist of little more than an embolus inside of a vessel, and which appear granular under low powers, but under high powers, especially after aniline staining, are found to be composed of bacteria. Older spots are surrounded by a hemorrhagic zone, and in still older ones suppuration has developed around the embolus. The more the suppuration spreads the more the real producers of the suppuration disappear, and in the larger abscesses it is no longer possible to demonstrate schizomycetes.

These processes have no clinical significance. It is unnecessary to say that they affect the function of the heart, but they cannot be diagnosed during life. The size of abscesses of the heart varies from that of a pin's head to that of a bean, a pigeon's egg, or even more. Roth reports a case in which the abscess contained 30 gm. of pus. Sometimes a number of abscesses are immediately adjacent to one another, or in direct communication, so that upon section through the heart we find a series of communicating cavities. Absorption of small abscesses is conceivable, and it may be assumed that they leave a fibrous cicatrix; this can hardly be looked for in large accumulations of pus. There are several possible terminations in such cases. In a series of cases the pus is converted into a cheesy, crumbly mass, in which calcification occasionally occurs. As a rule, a fibrous capsule forms at the periphery, so that the abscess is, to a certain extent, encysted. In other cases the abscess opens. If the abscess is near the pericardium, the pus will probably rupture into the pericardial cavity, giving rise to a diffuse, usually purulent, pericarditis, which, as a rule, proves fatal. Sometimes suppurative pericarditis occurs without rupture of the abscess, when the latter is situated immediately beneath the epicardium. In other cases the pus perforates into the cavities of the heart. The abscess continues to extend, in such cases, towards the endocardium, the superjacent layers of the endocardium become inflamed, and then the pus either breaks through the softened endocardium into the cavities of the heart, or the blood within the ventricle forces its way into the abscess cavity and clears it out. The immediate result of the mixture of pus with the blood within the heart is the production of a prolific source of emboli, inasmuch as plugs of pus corpuscles continue to be carried along in the circulation, until they stick in small vessels of the periphery. Emboli are observed most frequently in the spleen and kidneys, more rarely in the brain and arteries of the skin (giving rise to manifold pustular or hemorrhagic eruptions). If the rupture has occurred into the right ventricle, we may expect to find embolic foci within the lungs.

As soon as blood has entered and cleared out the abscess cavity, the remaining layers of muscle are often rapidly dilated, and the original abscess of the heart is converted into an acute cardiac aneurism. The dangers of the latter are extremely great, and the fatal termination often occurs in a short time. As the blood undermines the abscess walls more and more, the wall of the heart is progressively thinned, until it finally gives way, the heart ruptures, and sudden death occurs, with signs of internal hemorrhage and paralysis of the heart. Recovery from cardiac aneurism does not seem ever to occur, probably because the uninterrupted current of blood prevents every reparative process.

The perforation is sometimes preceded by the formation of long fistulous canals, so that the rupture occurs at a place remote from the site of abscess. It may then happen that the pus presses in between the endocardial layers of the venous valves, and thence effects its entrance into the ventricular cavity.

Noteworthy complications may arise if the abscess is situated in the upper part of the auricular septum. If the abscess ruptures, a direct communication may be formed between the right and left ventricles. But the perforation sometimes occurs on the right side superiorly and posteriorly, so that the left ventricle communicates with the right auricle. This is followed by marked dilatation of

the right auricle, inasmuch as a part of the left ventricular blood flows into the right auricle during systole. In certain cases the perforation occurs through anous, fistulous tracts, and these may be unattended with symptoms for a long time. If life is retained, the site of rupture not infrequently acquires a wall-like, ring-shaped rim of connective tissue, and thus prevents further changes. In other cases the perforation is attended with sudden changes in the valvular apparatus of the heart. Thus, the aortic valves may be drawn into the inflammatory process, partly destroyed, and soon made insufficient. The pulmonary valves or their sinus may also be destroyed, or there is a dissolution of those chordæ tendinæ of the tricuspid valves which spring from the ventricular septum, or the inflammatory process spreads directly to the leaflets of the tricuspid valve. These changes often produce insufficiency of the valves, and v. Bamberger shows that they may sometimes be utilized in making a diagnosis. For, if we detect sudden insufficiency of the aortic valve, followed rapidly by insufficiency of the pulmonary or tricuspid valves, we can hardly explain it in any other manner than by abscess of the ventricular septum. It is unnecessary to add that all these processes are a prolific source of embolism.

Chronic myocarditis is probably the only form which possesses clinical interest, because in certain cases it is susceptible of a probable diagnosis. Anatomically, it leads to the formation of connective tissue plates, heart callosities, hence the name *myocarditis interstitialis fibrosa s. productiva s. sclerosa*. The callosities are at first grayish-red, later grayish-white patches, streaks, and plates, which are scattered more or less profusely through the heart muscle. They contain, occasionally, pale yellow or brownish spots, which are found to consist of the remains of constricted and fatty muscular fibres, and muscle coloring matter.

The callosities are found most frequently in the wall of the left ventricle, particularly near the apex, and in the septum ventriculorum. As a rule, they occur in the right heart only during fetal life, and then often give rise to congenital heart disease. Their size varies extremely. In some places they merely constitute a broadening of the intermuscular connective tissue, in others they form radiating plaques which may reach the dimensions of a mark piece. They also vary greatly in thickness; not infrequently the whole thickness of the heart muscle is involved, so that the pericardium and endocardium are only separated by fibrous callosities. Occasionally they form firm, solid nodules. They may be so numerous that the larger part of the muscular tissue of the heart is replaced by plates of connective tissue.

They seem sometimes to follow an acute myocarditis, as is shown particularly by those fibrous cicatrices, in which thickened, cheesy, or calcareous pus is found. But there are also cases in which the process is chronic from the beginning, and always retains its interstitial character. Chronic myocarditis is associated usually with endocardial changes, which are shown by connective-tissue proliferation and thickening, and corresponding changes are also found on the epicardium. But it should be remembered that the endocarditic or pericarditic changes existed first, and that connective-tissue changes in the myocardium only developed secondarily. The heart muscle is often hypertrophic (*myocarditis hypertrophica sclerosa*), either as the result of valvular lesions or chronic nephritis, or of overwork, inasmuch as the heart muscle has lost in working material on account of the callosities, and the intact remainder must therefore unfold greater power. The changes in question are connected not infrequently with diseases of the coronary arteries, particularly with endarteritic changes.

The fibrous callosities are insignificant if present in small numbers, and of small dimensions. But if their number and extent are consider-



able, they must give rise to insufficiency of the heart's action, because the muscle has lost a portion of the material which was capable of function. But we must now consider other anatomical sequelæ.

Myocarditic development of callosities not infrequently affects the papillary muscles. They atrophy, and are converted, especially near the apex of the heart, into rigid, tendon-like structures. It is unnecessary to say that their function suffers in consequence, and as these changes are associated with thickening and retraction of the valves, it is evident that chronic myocarditis gives rise to valvular insufficiency.

Chronic myocarditis is also followed by that condition to which the term true stenosis of the heart has been applied by Dittrich. At times, the fibrous cicatrix is ring-shaped. This is found most frequently at the conus of the pulmonary artery, and, therefore, when the cicatrix retracts, it results in such marked stenosis of the beginning of the pulmonary artery that the same signs develop as in stenosis of the pulmonary orifice. These changes may develop during foetal life, but also occur at a later period, as was observed by Dittrich, in consequence of a blow. A similar condition has been found in the conus arteriosus, and this gives rise to the symptoms of aortic stenosis.

As the final result of chronic myocarditis, we must mention the development of chronic aneurism of the heart. It is not difficult to understand that the fibrous band in the heart constitutes a *locus minoris resistentiæ*. As the cicatrix is incapable of active contraction, it yields to the blood pressure in the cardiac cavities, and gradually folds outward into a sac-like cavity, the entrance to which is not infrequently very narrow and constricted. This dilatation is observed most frequently in the left ventricle near the apex (in fifty-five cases, or sixty-eight per cent, among eighty-seven cases collected by Pelvet). This author found only three cases in which the chronic aneurism was situated in the right ventricle, but cases have also been reported in which the auricle was the starting-point. Several aneurisms, usually in close proximity, are found occasionally in one heart (Thurnam described a case in which four were present). The aneurism may be as large as the heart, and Berthold described a case in which an aneurism, starting from the right auricle, was as large as a man's head, and extended from the left clavicle to the last true rib. It had partly eroded the ribs, and was situated immediately beneath the skin, through which drops of blood oozed. Arnott has described a cardiac aneurism, which started from the upper part of the left ventricle, surrounded the origin of the aorta, and embraced almost the entire heart. After cardiac aneurisms have reached a certain size, every trace of muscular tissue in their walls often disappears, and partial calcification occurs. There are very often adhesions between the outer surface of the aneurism and the parietal layer of the pericardium, and the aneurism may thus project into the left pleural cavity or be surrounded in great part by the left lung. Aneurisms situated on the ventricular septum almost always project into the right heart, on account of the greater blood pressure in the left heart. Rokitsansky observed projection into the left ventricle of an aneurism which developed during foetal life (greater blood pressure in the right ventricle in the foetus). The most frequent termination of chronic cardiac aneurism is rupture and rapid death. The hemorrhage occurs generally into the pericardium, but sometimes into the left pleura or even into the large arterial trunks.

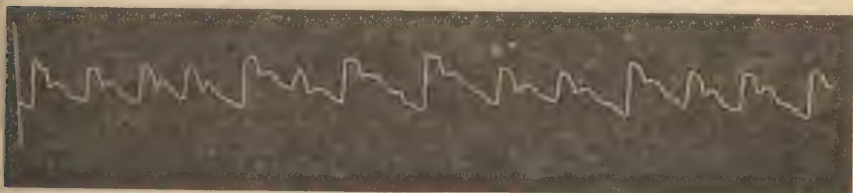
Caution must be exercised in the anatomical examination of the heart: successive horizontal incisions should be made through the organ, as this is the only way in which we can obtain a clear idea of the extent of the process.

II. ETIOLOGY.—Among the causes of chronic myocarditis are mentioned exposure, injury (fall, blow upon the chest), and bodily over-exertion. In many cases it is probably a senile change, especially when the disease is associated with endarteritic changes in the coronary arteries. Infectious diseases (acute articular rheumatism, malaria, syphilis) are often at fault. Sometimes the myocardium is directly affected, sometimes it is preceded by acute changes, which pass into a chronic stage, sometimes the chronic myocarditis depends on preceding pericarditis and endocarditis. Ruele states that chronic muscular rheumatism also predisposes to chronic myocarditis.

Toxic forms of myocarditis also occur. It is correctly held that drunkards often fall a victim to the disease. An injurious influence on the heart is also attributed, and not without justice, to the excessive use of tobacco, and to lead poisoning. Chronic myocarditis sometimes appears to be connected with diseases of nutrition, especially gout, and also with diabetes mellitus. It is not infrequently a concomitant of chronic nephritis, particularly of cirrhosis of the kidneys, in which affection it is dependent on endarteritic changes in the coronary arteries. It is also observed in conditions which have long been preceded by stasis of blood. The disease is more frequent in men than in women; it is rare in childhood, most common between the ages of twenty and forty years.

III. SYMPTOMS AND DIAGNOSIS.—The symptoms are often capable of such varied interpretation that the diagnosis is attended with great, often unconquerable, obstacles. The general effect is that the heart's action becomes more and more insufficient. The patients complain of palpitation of the heart and shortness of breath on slight exertion, and are, therefore, easily tired. During the attacks of palpitation, the patients may suffer from pains around the heart, radiating into the left arm, or the epigastrium. The apex beat, cardiac impulse, and heart sounds are remarkably feeble. The first ventricular sound is occasionally replaced by a systolic murmur. The heart's action is arrhythmical; an intermittent pulse is very often, a very slow pulse occasionally, observed. Sphygmographic examination shows that the individual beats are often of unequal vigor and duration. The patients have a tendency to

FIG. 10.



Pulse curve of the right radial artery of a man, æt. 52 years, suffering from chronic sclerotic myocarditis.

catarrh of the air passages, and the cervical veins are inordinately distended. The patients are generally cyanotic. Ruehle mentions the almost constant occurrence of digestive disturbances. If the symptoms of stasis gain the upper hand, œdema develops in the subcutaneous cellular tissue and the serous cavities, together with enlargement of the liver, bronchial catarrh, and hemorrhagic infarctions, and if the heart's action is not improved, death occurs from pulmonary œdema, pneumonia, hamoptysis, or cerebral congestion. I have repeatedly observed sudden death occur, although the autopsy afforded no satisfactory explanation.

The recognition of a chronic cardiac aneurism is also attended with great difficulties, and can rarely be effected with any degree of probability. Aran stated that, in aneurisms situated near the apex, the cardiac impulse is absent over the lower part of the heart, and is present only over the upper part, but this statement is more the result of theoretical considerations than of practical experience. In some cases, we have to deal with a pulsating tumor, and must, therefore, be careful not to mistake it for an aortic aneurism. In the majority of cases there

is an entire absence of symptoms, for we will rarely diagnose an aneurism of the heart from enlargement of the area of cardiac dulness.

IV. PROGNOSIS.—The prognosis is always grave, for even if we succeed in relieving the symptoms of cardiac insufficiency, the danger is only averted temporarily. Ruehle states that the diuresis is a prognostic standard, inasmuch as there is hope of therapeutic success so long as we can succeed in increasing the excretion of the urine. The prognosis is relatively favorable in syphilitic cases, as suitable therapeutic measures may effect resolution of the myocarditic changes.

V. TREATMENT.—Absolute bodily and mental rest, and an easily digested, but nutritious diet, are the chief measures of treatment. If the heart's action is accelerated and irregular, an ice bag may be applied over the organ, and retained for a long time. The symptoms of sudden heart failure or chronic insufficiency of the heart's action are treated in a similar manner to fatty heart (vide page 58).

If syphilis is suspected as the cause, we should prescribe iodide of potassium (10 : 200, one tablespoonful t. i. d. an hour after meals), and mercurial preparations (ung. hydrarg. cin., 5.0 daily by inunction; hydrarg. jodat. flavi 0.5 : pulv. et succ. liq. q. s. ut f. pil No. 50. D. S. one pill t. i. d. after meals).

#### 6. *Spontaneous Rupture of the Heart. Cardiorkesis Spontanea.*

I. ETIOLOGY.—Spontaneous rupture of the heart only occurs when there have been previous changes in the muscular fibres which have impaired their power of resistance. According to some of the older observations, the unchanged heart may rupture spontaneously, but this is doubted. At all events, nothing similar has been reported in recent times, since microscopical examinations have been made.

Cardiac rupture occurs most frequently in fatty heart. Among 83 cases of fatty heart, collected by Quain, sudden death from rupture of the organ occurred in 28 (34 per cent). Myocarditis is also an etiological factor. Rupture of the heart results not infrequently from cardiac abscesses and acute and chronic aneurisms which have been produced by myocarditis. In certain cases, circumscribed dry pericarditis may lead to local fatty degeneration of the heart muscle, and to rupture of the organ.

Among affections of the valves, orifices, and main trunks, stenosis in particular may lead to rupture of the heart if it has been preceded by fatty changes in the heart muscle. Stenosis of the aorta at the site of insertion of the ductus Botalli is most important in this particular.

Diseases of the coronary arteries must also be mentioned. Richard describes an aneurism of the coronary artery which led to erosion and rupture of the heart muscle. In other cases, embolic or thrombotic plugging of these vessels has given rise to abscesses, or softening of the heart muscle (myomalacia cordis). Finally, neoplasms and echinococci in the heart muscle may cause rupture of the organ.

Rupture of the heart may occur unexpectedly during complete mental and bodily rest, and in a number of cases the accident occurred during quiet sleep. In other cases some mental or bodily exertion is the immediate cause. Thus, it has been observed after lifting a heavy load, after dancing, after a heavy meal, during defecation, coitus, an epileptic convulsion, etc. Tenison recently reported a case of cardiac rupture



during the act of vomiting, and v. Buhl observed it during a coughing spell. Franz reports rupture of the heart during delivery; Spiegelberg, during the puerperal condition. It may also occur during a cold bath, inasmuch as the contraction of the cutaneous vessels presents an abnormally great resistance to the left ventricle. Changes in the weather, accordingly, may also exert some influence. Rupture of the heart has been observed occasionally after a trivial injury, and this possibility may be important from a medico-legal standpoint. An historical case is usually chosen by the text-books to show the effect of mental excitement, viz., that Philip V. of Spain died suddenly of rupture of the heart on receiving the report of the defeat at Piacenza.

It has been disputed whether the rupture occurs during systole or diastole. It will evidently occur with the greatest probability when the pressure within the cardiac cavities is greatest. As Wunderlich remarks, this is the case at the beginning of systole.

Rupture of the heart occurs more frequently in men than in women. It is a disease of old age, as it occurs generally after the age of sixty.

II. ANATOMICAL CHANGES.—From an anatomical standpoint we differentiate total and partial rupture of the heart. In the former there is a solution of continuity throughout the entire thickness of the heart walls, in the latter a solution of continuity of individual layers of muscle, individual trabeculæ, or papillæ.

Total rupture occurs most frequently in the left ventricle, because the antecedent muscular changes develop by preference in the left ventricle. It is most common on the anterior wall of the ventricle near the apex and extends not infrequently to the lowest portion of the ventricular septum. The posterior ventricular wall is rarely the site of rupture. The site of rupture is also relatively frequent in the right ventricle, more rare in the septum ventriculorum than in the right auricle, most rare in the left auricle.

The endocardial and epicardial openings at the site of rupture are usually irregular and jagged. Sometimes the one is larger, sometimes the other; they are often plugged by clots of blood. The length of the rupture rarely exceeds one cm., but cases are known in which it extended the whole length of the ventricle. The rent follows the course of the muscular fibres and layers, for which reason it forms a sinuous, almost fistulous tract within the heart muscle, so that the internal and external openings are not opposite one another. The rent rarely occurs transversely through the muscular fibres, and such cases are regarded as especially dangerous. In the auricles the muscular fibres may not be torn, but merely separated from one another.

Usually there is but one rent, but it may happen that the originally single canal bifurcates peripherally, and opens into a number of epicardial fissures. From this class must be distinguished those cases in which the epicardium forms a sort of bridge over the site of rupture. Andral had observed, however, that five distinct points of rupture occurred in one heart, and recently Barclay and Paget described several openings in the right ventricle.

The muscular layers immediately adjacent to the site of rupture are undermined, particularly on the endocardial side, disintegrated, and more or less infiltrated with blood. The blood escapes almost always into the pericardial cavity. Sometimes an extraordinary amount escapes at once, sometimes there is a gradual oozing. The blood in the pericardium may amount to several pounds, and, on removing the sternum, the

pericardium then appears as a tense, large, fluctuating, bluish-black cyst. If pericardial adhesions are present, as occurs particularly in chronic aneurism of the heart, the hemorrhage may occur into the pleural cavity, and even into the large arterial trunks.

Old fibrous cicatrices are found occasionally in a fresh rupture. Some authors suppose that this indicates a possible recovery of a rupture in rare cases. But not all the observations in question are authentic, and indeed it is not very probable that a markedly fatty heart muscle presents a great tendency to the formation of a cicatrix.

Partial rupture of the heart muscle, when the papillary muscles are torn and the chordæ tendineæ are ruptured, gives rise to sudden insufficiency of the venous valves.

III. SYMPTOMS.—Death from total rupture of the heart not infrequently occurs suddenly and unexpectedly in individuals who have been regarded as healthy.

In other cases the catastrophe is delayed for a few hours. The patients not infrequently cry out that something has been torn around the heart. Indefinable pain and terror overpower them and distort their features. The face grows pale, the skin cool, the face and skin are covered with cold, clammy sweat, the pulse is extremely small and rapid. Other signs of internal hemorrhage are very soon superadded. Among these are violent vomiting, sometimes associated with severe diarrhœa, so that the symptomatology somewhat resembles that of cholera. Some attribute the vomiting to anæmia of the brain and irritation of the pneumogastric, others assume a mechanical irritation of the fibres of the pneumogastric, by the blood which has escaped into the pericardium. The increasing anæmia of the brain is shown by syncopal attacks, spots before the eyes, ringing in the ears, and perhaps convulsions immediately before death.

It is readily understood that examination of the heart is especially important. The apex beat usually grows feeble and imperceptible; the heart sounds are extremely weak, but occasionally there are vigorous, irregular contractions of the heart. The demonstration of a rapidly increasing enlargement of cardiac dulness produced by the extravasation of blood is extremely important.

Death ensues generally in a few hours, in rare cases it is delayed until the lapse of a few days. May reports one case in which it occurred on the seventeenth day. If the hemorrhage is sudden and profuse, the cardiac movements are interfered with to such an extent that death results from paralysis of the heart. For, although we may find a large amount of pericarditic fluid in pericarditis without complete inhibition of the movements of the heart, it must not be forgotten that there is a gradual accumulation of fluid in such cases. In some cases death is the result of cerebral anæmia. Cases remain, however, in which the amount of blood extravasated is too slight to interfere with the movements of the heart, or to produce cerebral anæmia. We are then compelled to assume certain disturbances of innervation, known under the term shock.

Partial rupture of the heart may be diagnosed when it affects the papillary muscles and has given rise to rupture of the chordæ tendineæ. This gives rise to symptoms of sudden insufficiency of the venous heart valves. In many cases its development is accompanied by an unspeakable pain in the region of the heart, and death may follow very rapidly.

In other cases compensation of the circulatory disturbances is conceivable by the aid of secondary dilatation and hypertrophy of the heart.

IV. DIAGNOSIS.—The diagnosis can rarely be made with certainty. In total rupture it is only possible when signs of internal hemorrhage, increasing heart failure, and rapid increase of cardiac dulness are associated with one another. In many cases we must be satisfied with a probable diagnosis, based more on experience than on objective signs.

V. PROGNOSIS.—The prognosis is unfavorable; it is very doubtful whether recovery is even possible.

VI. TREATMENT.—In those patients in whom we may assume the existence of fatty heart, treatment must be directed to prophylactic measures. These refer particularly to the avoidance of bodily and mental exertion.

If we are justified in assuming the occurrence of rupture of the heart, we should make subcutaneous injections of ergotin (ergotinina Bombellon, one-half syringe-ful with an equal amount of water), and apply an ice-bag over the heart to prevent the further extravasation of blood as much as possible. Wine, ether, camphor, musk, etc., may be given to prevent dangerous exhaustion (vide the recommendations on page 58). Under certain circumstances, digitalis in large doses (2 : 100, a table-spoonful every hour) may prove useful by slowing the action of the heart.

#### 7. Tumors of the Heart. *Neoplasmata Cordis.*

1. Tumors of the heart muscle are rare, can hardly ever be diagnosed during life, and therefore possess slight practical importance. Indeed, tumors of small dimensions give rise to no disturbances. Larger ones cause insufficiency of the heart's action by compressing and destroying the muscular fibres. But, occasionally, they project into a cardiac orifice to such an extent that they first narrow and then occlude it altogether, and thus prove fatal. They may also prevent the unfolding of the valves, and thus give rise to symptoms of valvular insufficiency. Or parts of the new growth break off, and are carried into peripheral arteries—brain, extremities, lungs, etc.—followed by the symptoms of embolism. Sudden death results, as a rule, if the origin of the aorta or pulmonary artery is occluded by an embolus. The symptoms referred to are so manifold that we do not gain a clear idea during life, even if tumors, which may be regarded as the starting-point for metastases in the heart, can be detected in peripheral organs.

Cancer, sarcoma, leukæmic new-formations, tubercle, gumma, myxoma, lipoma, fibroma, and myoma have been observed in the heart.

#### 8. Parasites of the Heart Muscle.

1. The animal parasites which may be found in the heart are *cysticercus cellulosæ*, *pentastomum denticulatum*, and *echinococcus*. The two former possess no practical significance, the latter very little.

According to Griesinger, *echinococcus* vesicles are more frequent in the right than in the left ventricle. They are either present in the heart alone, or—and this is the rule—are associated with *echinococci* in other organs, generally the liver. Their size varies from that of a pin's head to that of an apple. Their number is occasionally very considerable. In one case Otto counted eighty vesicles in the heart. The sac very often ruptures, and the individual vesicles enter the current of



blood. Among twenty-one cases collected by Oesterlen, sudden rupture occurred six times (nearly twenty-nine per cent). Sudden death may occur if the vesicles occlude a cardiac orifice, and also if the trunk of the pulmonary artery or one of its main branches is occluded. In other cases the lungs are flooded with echinococcus vesicles, and in Barclay's case the patient expectorated the vesicles. In this case, one vesicle burst into the pleura, and gave rise to pleurisy. Very grave disturbances ensue if the vesicles enter the left cardiac cavities. Oesterlen mentions a case in which gangrene of a limb, necessitating amputation, suddenly developed; death occurred from pyæmia. The cause of the gangrene was occlusion of the right common iliac by echinococcus vesicles starting from a cyst in the posterior wall of the left ventricle, and projecting into the left auricle, and, at the same time, into the pericardial cavity.

## PART III.

### DISEASES OF THE ENDOCARDIUM.

#### 1. *Inflammation of the Endocardium. Endocarditis.*

Modern experience has shown that endocarditis is a frequent disease. It almost always develops in localized patches, and is rarely diffuse. According to its localization, we distinguish endocarditis valvularis, chordalis, papillaris, trabecularis, parietalis. Special clinical importance attaches to endocarditis valvularis because in the majority of cases it is the origin of chronic valvular affections of the heart (insufficiency or stenosis). In the large majority of cases, endocarditis develops in the left heart; it is observed more frequently on the right side during foetal life. The reasons for this are unknown, though two factors may be taken into consideration. In the first place, the right ventricle of the foetus contains the blood which has been arterIALIZED in the placenta, and in the second place greater work devolves upon the right side of the heart during foetal life.

Endocarditis may be acute, subacute, or chronic. Acute endocarditis is characterized by a tendency to destruction of the inflamed tissue, and to the formation of ulcers. We will term this form acute septic endocarditis, as it generally presents symptoms of severe septic general infection. In subacute endocarditis, warty outgrowths and proliferations develop at the site of inflammation (endocarditis verrucosa of Virchow). In chronic endocarditis, there is a striking tendency to retraction of the endocardial tissue (endocarditis chronica retrahens).

The tendency of recent opinion is to attribute acute and subacute endocarditis to the influence of certain bacteria. The demonstration of schizomycetes has generally been successful in acute septic endocarditis. Among fourteen cases of endocarditis, Hamburg recently found schizomycetes upon the inflamed places in only four cases, but Koester has shown that, apart from the technical difficulties, the results of the examination may be rendered negative by the involuntary removal of the bacteria in the ordinary method of post-mortem examination (introduction of the fingers into the cardiac orifices of the unopened heart, in order to recognize stenosis, or insufficiency). If Klebs' investigations are substantiated, there are special forms of bacteria in acute, and in subacute endocarditis. Klebs distinguishes the first form as septic endocarditis, the latter as rheumatic or monadetic endocarditis. He finds the micrococci of the latter larger than in the former; they do not have a brownish color, are arranged in parallel rows, and imbedded in a jelly-like mass which forms a bright zone around each granule.

### a. *Acute Septic Endocarditis.*

(*Endocarditis ulcerosa, maligna, diphtheroides. Mycosis Endocardii. Necrotic, acute destroying, infectious, mykotic, bacteritic endocarditis.*)

I. ETIOLOGY.—Virchow first discussed the disease in detail as the result of puerperal disease, and believed that it must be attributed to parasitic influences. Micrococci were first demonstrated positively by Winge and Hjalmar Heiberg. The bacteria generally effect an entrance into the body through a wound, so that acute septic endocarditis is generally a secondary affection.

Very slight wounds often are sufficient: for example, opening a suppurating corn, a small furuncle, frost bites, even scratch marks, and superficial erosions of the skin. A frequent source of septic endocarditis is puerperal fever, in which imperfect development of the vascular apparatus (hypoplasia) gives rise to an undoubted predisposition to the disease (Virchow).

Acute septic endocarditis occurs next after infectious diseases (acute articular rheumatism, scarlatina, and other acute infectious exanthemata, diphtheria, typhoid fever, periostitis, and osteomyelitis). According to Lancereaux, it occurs with special frequency under the influence of malaria. Wilks described a case following empyema, Orth one following pyelo-nephritis.

Finally, cases remain in which no cause can be demonstrated (idiopathic endocarditis). This category includes those cases in which the patients attribute the disease to over-exertion and sudden cooling of the perspiring body. Under such circumstances, it is assumed that the infecting schizomycetes enter the blood from the air passages, or intestinal tract without giving rise to any changes at the point of entrance.

The disease is more frequent in women than in men, on account of the frequent development of the puerperal form of affection. For this reason, also, it is most common from the age of twenty to forty years. It may also develop during childhood (Cayley describes a case in a boy of nine years).

We may here refer to a striking peculiarity of the disease. In reading the clinical histories, we are struck by the frequent mention of an antecedent articular rheumatism, and the frequent observation of old endocarditic changes in addition to the fresh septic processes. It would seem, therefore, as if such processes furnish an especially suitable opportunity for the deposit and proliferation of the bacteria circulating in the blood. According to Virchow, hypoplastic errors of development in the vascular apparatus appear to act in the same way. But Koester believes that the organisms do not pass inwards from the free surface of the endocardium, but that they follow the opposite course. He believes that, through the agency of the coronary vessels, embolic occlusion with bacteria occurs in the endocardial blood-vessels, and that thence they pass into the tissue and to the free surface of the endocardium. Recent investigations into the distribution of the blood-vessels in the valves are not very favorable to this view.

II. ANATOMICAL CHANGES.—Septic endocarditis involves most frequently the left ventricle, perhaps because the endocardium here is apt to present the changes which are preparatory to the deposit and proliferation of the bacteria. A number of cases have been reported, however, in which the right heart was alone affected. The tricuspid or pulmonary valves may be affected separately or in combination.

Septic endocarditis also corresponds to the other forms with regard to the seat of the process. Endocarditis valvularis is much more frequent

than parietal endocarditis. The former develops preferably at the lines of closure of the valves, particularly in those places which are subjected to the greatest friction and mechanical irritation by the current of blood. The semilunar valves are affected, accordingly, upon their ventricular aspect, while the mitral valve is generally affected on its auricular surface. The attempt has been made to explain this fact on the ground that the bacteria are here, to a certain extent, pressed from the blood between the endothelium cells and then into the tissue proper of the endocardium.

The lines of closure of the valves can be readily recognized. They are those lines along which the valves, during closure, are in direct contact. In the semilunar valves, the lines of closure touch the free borders only at the insertion of the leaflets, and at the nodulus Arantii, and at other places are 1-2 mm. distant from the free edges of the valves. In the mitral valve, the line of closure is distant about 1 mm. from the free edge of the valve. Upon the auricular surface of the valve, it corresponds exactly to those points at which, upon the ventricular aspect, the chordæ tendineæ are inserted.

The first changes in septic endocarditis consist of dull shining, velvety, finely-granular deposits on the endocardium, looking as if they had been breathed upon. If they are removed by scraping, a superficial defect in the endocardium becomes visible. This is usually sharply defined, jagged, and its base is covered with a grayish-red or yellow coating, while the edges are injected and slightly elevated.

Where the process is farther advanced, larger thrombotic deposits are found on the endocardium, consisting of brittle, brownish-red or grayish-red masses as large as a cherry, or even more. Upon removing them, a more or less deep loss of substance makes its appearance. Excreescences and proliferations are found not infrequently in the vicinity. In addition, there are usually older changes in the endocardium; thickening, fatty changes, calcification, retraction.

There is a very noticeable tendency of the process to destruction of tissue and ulceration, giving rise to very important sequelæ. If a lamella of the endocardium has been destroyed in one part of the valvular apparatus, the others are not infrequently dilated by the current of blood, giving rise to an acute aneurism of the valve. This forms a sort of rounded sac, which is adherent to the valve by a narrow, neck-like aperture. It may attain the size of a walnut, and contains fluid blood or older thrombi. The direction taken by the aneurism may be foretold. Upon the semilunar valves the aneurism looks into the ventricular cavity, while on the mitral valve it is directed towards the auricle. This is the natural result of the function performed by these valves in the circulation. If the wall of the aneurism is no longer capable of resisting the blood pressure, it bursts, and insufficiency of the valve results.

But sometimes the valvular insufficiency occurs in another manner. On the mitral valve, it may happen that the inflammatory process extends to the chordæ tendineæ, destroys them, and thus frees the lower rim of the valve, so that at each systole it falls back unhindered into the auricle. On the semilunar valves, the ulceration extends occasionally to the point of insertion of the valves, separates it, and thus insures the possibility that, during ventricular diastole, the blood flows from the aorta or pulmonary artery into the left or right ventricle.

In endocarditis parietalis, which occasionally occurs secondarily because, during the movements of the valves, the inflamed portions of the valves are momentarily in contact with the parietal endocardium, and in-



fect its surface with bacteria, inflammation and degeneration of the heart muscle may ensue, with the formation of an acute cardiac ulcer. Sometimes the destruction of the heart muscle extends so far that an abnormal communication is formed between the cardiac cavities. It may also happen that the inflammation crawls along between the two endocardial layers of the valves to the heart muscle, and there gives rise to the above-mentioned changes.

Virchow showed that the base of the ulcerations and the deposits upon them consisted of a finely granular substance, very similar to necrotic (diphtheritic) masses. By the aid of high powers and suitable reagents it has been found that these consist of partly round, partly rod-shaped bacteria. The round bacteria are found either isolated among the rod-shaped ones, or in groups, or arranged in chains. Sometimes they far exceed the rod-shaped ones in number. Probably we do not have to deal with different stages of development of one form of schizomycetes, but various forms of bacteria are capable of producing acute septic endocarditis. The bacteria are distinguished from granular detritus by their resistance to acids, alkalies, boiling, and treatment with alcohol, ether, chloroform, and benzin, and their bright staining in aniline colors. Some of these reactions also prove that they are not fatty granules.

In many cases, the tissue proper of the endocardium presents extremely slight changes. Sometimes we find only a slight swelling of the tissues, or necrosis and disappearance of the cellular elements. In other cases, there are undoubted signs of inflammation, numerous round cells being found in the superficial layers of the endocardium, so that it looks almost like granulation tissue under the microscope. In one case Koester observed emboli of masses of bacteria in the vessels of the endocardium; dilatation of the blood-vessels has also been described. Distention of the lymph-spaces with bacteria has also been noticed in some cases.

Eberth first showed, experimentally, that the mykotic masses possess a high degree of infectiousness; inoculation into the cornea of the rabbit was soon followed by opacity and necrotic destruction. This experiment explains why so many organs are affected secondarily, and the disease runs its course as a very severe general infection.

There is hardly an organ which has not been found affected secondarily in acute septic endocarditis. The infection is effected through the blood-vessels, the current sweeping off the bacteria from the endocardium and depositing them in other organs as emboli and producers of inflammation.

These mykotic emboli are found in the heart muscle itself, constituting miliary, rounded, pale gray spots as large as a pin's head, surrounded generally by a hemorrhagic zone. Their number may be extremely large. Consisting at first of nothing but a mykotic embolus, they may afterwards become converted into abscesses by giving rise to inflammatory processes (vide page 60). Bacterial emboli are observed with special frequency in the spleen and kidneys. In the latter they are found partly in the cortex, partly in the papillæ, in which they give rise not infrequently to gray, streaked, closely aggregated markings. Under the microscope it is found that in the cortex they have entered the vessels of the glomeruli, whence they pass into the tubules and give rise, in part, to the previously-mentioned changes in the papillæ.

Similar emboli have also been observed in the liver, gastro-intestinal mucous membrane, mesenteric glands, mucous membrane of the urinary passages, testicles and seminal vesicles, medulla of the bones, thyroid

gland, mucous membrane of the air-passages, the buccal and pharyngeal cavities, retina, choroid and iris, brain, serous membranes, skin, voluntary muscles, and diaphragm; in short, in almost all organs and tissues.

The changes in these various organs are similar to those in the heart: at first mechanical embolic occlusion, later, inflammation, abscess formation, and ulceration. Thus Edler describes such ulcerations upon the mucous membrane of the larynx and trachea, and similar changes have been observed in the intestinal mucous membrane. In the serous cavities purulent inflammation develops, so that suppuration occurs in the joints, the pleural, pericardial and peritoneal cavities, and the meningeal spaces. A very peculiar clinical history may be produced in this manner; Harme describes a case which ran a fatal course with the symptoms of cerebro-spinal meningitis. Bacteria have been demonstrated repeatedly in the purulent exudations. Lehman and van Deventer observed schizomycetes, partly free, partly inclosed in pus cells, in a purulent pleuritic exudation. Purulent processes and destruction of the eyeball have been repeatedly observed.

Sometimes miliary emboli do not develop exclusively; if larger pieces of the endocardium are separated by ulceration and carried into the blood-current, they give rise to gross emboli which will cause severe nervous symptoms if they lodge in the cerebral vessels. Paralysis which, if the result of miliary emboli, can only occur when the emboli are very numerous and situated close together, develops not infrequently during the course of septic endocarditis. Cases have been reported in which hemiplegia developed unexpectedly, was followed rapidly by death, and resulted from a slowly-developed endocarditis. Miliary mykotic foci may be suspected when the paralysis is confined to one nerve (very often the facial nerve), or paresis alone is produced, while marked hemiplegia favors the assumption of gross emboli.

Since septic endocarditis often develops after preceding verrucose endocarditis, it is not surprising that old embolic occlusions, particularly the wedge-shaped infarctions of the spleen and kidneys, are found not infrequently in addition to the recent emboli. The internal organs occasionally contain larger abscesses, the result of suppuration of old wedge-shaped infarctions.

But we must not look upon all extravasations of blood in the internal organs, upon the mucous and serous membranes and the skin, as the result of capillary mykotic emboli. Not infrequently there are signs of dissolution of the blood, as a result of which the vascular walls become abnormally permeable to the red blood-globules. These may be mistaken so much the more readily for emboli because, as Litten showed with regard to retinal hemorrhages, even the pure extravasations of blood present a bright, whitish-yellow centre, but they do not contain bacteria.

Many authors have called attention to the dark lac color of the blood, which is often, also, extremely thin.

Virchow noticed, in one case, an acid reaction of the blood on the second day after death, and after removal of the albumin, leucin and tyrosin were deposited.

The remaining changes in the internal organs are those of severe febrile general infection, viz.: cloudy swelling or fatty degeneration. The latter is apt to be particularly well marked in the heart muscle, and this may be the immediate cause of death in certain cases. Wax-like

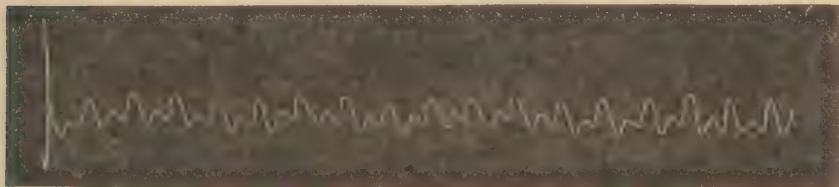
degeneration of the heart muscle also occurs. In some cases the heart muscle has been found intact, but the disease then appears to have run a very rapid course. Enlargement of the spleen, which is hardly ever absent, must also be attributed to the general infection. The enlarged spleen is generally soft and diffuent.

A part of the changes visible during life disappear after death, particularly the roseolar and erythematous changes of the skin, while the pustules, pemphigoid vesicles, petechiæ, and ulcerations persist.

III. SYMPTOMS.—The symptoms are extremely manifold on account of the implication of so many organs. In some cases death occurs in a few hours or days, preceded for a short time by paralysis and severe cerebral disturbances. Sioli describes a case which ran a fatal course with symptoms of delirium acutum, and had been sent to an insane asylum. In other cases the causes of the septic endocarditis are so prominent that the latter remains hidden, and is only recognized at the autopsy. But even when the symptomatology approximates our paradigm, there are so many deviations that we may boldly maintain that no two cases are entirely alike. According to the most prominent symptoms, we speak of septic endocarditis with a typhoid-like or an intermittent-like course.

In septic endocarditis of a typhoid character, the patients create an

FIG. 11.



Excessively dicrotic pulse of the right radial artery in acute septic endocarditis. Typhoid course. Axillary temperature 38.5° C.

impression as if they were suffering from severe typhoid fever. They have high fever, are apathetic, often delirious, and lie with half-closed eyes. The pulse is accelerated, full, soft, and dicrotic (vide Fig. 11); tongue dry, grayish-yellow or brown, not infrequently very red at the tip; later, often fissured, bloody, and fuliginous. The abdomen is tympanitic. Demonstrable enlargement of the spleen, diarrhoea (occasionally bloody), and roseola point distinctly to typhoid fever. Changes in the heart may not be recognized. Dilatation of the right heart (extension of greater [relative] cardiac dulness beyond the right edge of the sternum), or systolic murmurs at the apex, or at all the cardiac orifices, are sometimes present, but they are also observed not infrequently in typhoid fever. Days, and even weeks may elapse before the diagnosis is decided. In other cases the diagnosis of typhoid fever, which had been made with certainty, is suddenly overthrown on account of new developments, and changed to septic endocarditis.

Special diagnostic importance attaches to the embolic processes. But, as miliary emboli in the internal organs are, in part, unattended with symptoms, we must direct our attention to embolic changes in the skin, mucous membrane of the mouth, and retina. Upon the skin, the emboli appear as extravasations of blood, with a bright-yellow centre, the



embolus proper. Pustular and pemphigoid eruptions also appear to be connected with embolic processes in the skin. Under certain circumstances, circumscribed or even extensive gangrene of the skin is produced.

Bouchut describes a case in which suppuration occurred in the nail-bed of the fingers.

Other cutaneous affections are also observed, sometimes roseola-like, measles-like, scarlatina-like changes; sometimes erythematous and erysipelatous changes. Occasionally there are extensive hemorrhages into the skin, forming under the eye of the observer, enlarging and coalescing. Miliaria develops if there is profuse perspiration. Numerous small hemorrhages, whose colorless centre appears to demonstrate their embolic character, also develop on the mucous membrane of the lips and cheeks, the gums, tongue, and soft palate.

In one case I saw these lesions develop into shallow ulcers of the buccal mucous membrane. In another, repeated profuse epistaxis occurred, and was checked with difficulty.

Examination of the eye is very important in diagnosis. Extravasations beneath the conjunctiva are often observed as the result of embolic occlusion of the subconjunctival vessels. They are observed very constantly on ophthalmoscopic examination of the retina. The intimate relation between the retinal vessels and their white centre demonstrates their origin. Hemorrhages also occur remote from the retinal vessels, independently of embolic processes but readily mistaken for them, because they also often contain a brighter centre. These hemorrhages may develop in a very short time, and we sometimes find that the previously intact retina is strewn with hemorrhages after the lapse of a few hours. We must finally mention the Roth patches, which appear in ophthalmoscopic examination as whitish-yellow patches in the retina. Panophthalmitis purulenta may be associated with the other changes.

A valuable aid in diagnosis is afforded by pareses and paralyses which, combined with the previously-mentioned symptoms, point to the existence of a focus from which the organism is more and more flooded with emboli.

Inflammations of the serous membranes or pneumonic processes have a tendency to confuse the clinical history. Especially in those cases in which the disease has not been followed from the start, we can hardly avoid mistaking for a severe infectious pneumonia, a meningitis, pleurisy, or pericarditis, what is really the result of a hidden septic endocarditis.

The urine often contains albumin, more rarely blood. Martini's statements concerning the presence of bacteria upon renal casts require careful and strict control.

The fatal termination occurs sometimes with signs of severe general infection, sometimes with symptoms of heart failure, sometimes with grave complications on the part of the nervous system (paralyses and spasms) or the respiratory tract. Post-mortem rise of temperature of 43° C. has been repeatedly observed.

Violent vomiting and obstinate diarrhoea sometimes occur at the beginning of the disease, so that we are reminded of Asiatic cholera. This choleric form of endocarditis may prove fatal very rapidly with symptoms of

collapse. In other patients icteric symptoms appear. Sometimes they seem to result from slight catarrh of the ductus choledochus, sometimes from severe infection of the blood (hæmatogenous icterus), the blood-corpuscles being dissolved within the vessels as the result of the general infection, the coloring matter, which is set free, being converted into biliary coloring matter. The resulting symptomatology may be exactly like that of acute yellow atrophy of the liver.

Septic endocarditis of the intermittent type resembles intermittent fever on superficial observation. The main symptoms are enlargement of the spleen, and chills followed by fever. The fever (lasting a few hours) not infrequently terminates exactly as in intermittent fever, with sweating which often gives rise to a profuse eruption of miliaria, while the chills may recur at the same time, following the quotidian, tertian, or quartan type. Outside of the febrile period the patients may feel comparatively well. They are usually extremely pale, suffer from anorexia, but often ask for weeks for permission to leave the bed, without entertaining a suspicion of their dangerous foe. Examination of the heart furnishes no definite results. It is either entirely negative, or we detect slight dilatation of the right heart and a systolic murmur over the cardiac orifices.

The differential diagnosis between intermittent fever and septic endocarditis may be undecided for a long time, particularly if the endocarditis is primary. The case is suspicious if defervescence, as so often happens, is not complete, or the occurrence of the chills grows more and more irregular. In other cases pain in the region of the kidneys and hæmaturia point to embolic changes in the kidneys, and draw our attention to septic endocarditis. The cardiac phenomena must also be carefully followed. Sometimes the systolic murmur at the apex grows louder and louder, the dilatation of the right ventricle increases, the diastolic sound over the pulmonary artery has become very clicking, in other words, the signs of mitral insufficiency have developed under the eyes of the observer. The phenomena become still less doubtful if diastolic murmurs develop alone or in combination with the systolic murmurs.

Strict intermittence of the fever may persist several weeks, but gradually it grows more and more continued. The patients lose strength. The changes described in the preceding form appear upon the skin, mucous membranes, and fundus of the eye; paralysis or convulsions are observed not infrequently. Towards the end of life the intermittent type may change entirely to the typhoid type.

The disease often lasts a number of weeks. In a case belonging to the intermittent type, and in which I verified the diagnosis on autopsy, death occurred in the beginning of the eighth week. In certain cases the disease attacks the victim unnoticed, and proves suddenly fatal within a few hours or days.

IV. DIAGNOSIS.—As a rule, the disease is recognized with great difficulty. We must not depend upon a certain symptom, but draw our conclusions from the course of the disease and the ensemble of symptoms. Special weight must be attached to embolic processes, which are susceptible of external examination. Litten mentions that retinal hemorrhages may be employed in the differential diagnosis of typhoid fever and septic endocarditis, although he goes too far when he denies entirely the occurrence of retinal hemorrhages in typhoid fever. The chief remaining points in diagnosis have been previously referred to.

V. PROGNOSIS.—The prognosis is unfavorable and, as a rule, death is unavoidable.

VI. TREATMENT.—The objects of treatment are to maintain the strength, to destroy the bacteria, combat the inflammation, and prevent detachment from the endocardium and the production of emboli.

To maintain the vital energies we should recommend a rational diet: milk, eggs, beef tea, wine, and alcoholics. Dangerous conditions of collapse are combated by the administration of ether, ammonia, valerian, camphor, musk, etc. Subcutaneous injections of camphor dissolved in oil of almonds (1 : 10, a syringe-ful subcutaneously) may be given to patients who are unable to swallow.

To destroy the bacteria, we give internally salicylic acid or salicylate of sodium (0.5 every hour), together with large doses of alcohol, in order to prevent the prostration which is increased and in part produced by profuse perspiration. If the patient is very feeble, it is better to give quinine. Corrosive sublimate has been sometimes employed, ostensibly with success. In a woman who was recently sent into the Zurich Hospital (with the wrong diagnosis of typhoid fever), at the beginning of the fourth month of the disease, I saw very rapid recovery after the use of the following prescription:

R Quin. muriat.....5.0  
Hydrarg. bichlor. corrosiv.....0.2  
Pulv. Althææ, q.s. ut f. pil. No. 20.  
D. S. 1 Pill t. i. d. after meals.

Benzoic acid and benzoate of sodium deserve consideration, but not much can be expected from resorcin on account of its collapse-producing effect. But these remedies are also antipyretics, and, as the heart's action is thereby slowed, we cannot deny a certain effect in diminishing inflammation.

The inflammation is improved to a considerable degree by the constant application of an ice-bag to the region of the heart, and if the heart's action is too rapid, the cautious use of digitalis and nitre seems to be indicated:

R Inf. fol. digit..... 2.0 : 180.0  
Kali nitric .....10.0  
Syrup simpl.....20.0  
M. D. S. One tablespoonful every two hours.

Sedation of the heart's action is also the best method of preventing embolic processes.

The manifold complications of the disease must be treated according to well-known rules.

### *b. Endocarditis Verrucosa.*

*(E. villosa s. papillaris.)*

I. ETIOLOGY.—This form of endocarditis is either primary or secondary. The primary form includes those cases which result from injury. Chvostek recently described a case of this kind. Recent endocarditis of the pulmonary valves was found in a soldier who fell from his horse, and died six days later. Sudden exposure has also been regarded as a cause. v. Dusch and Charon observed acute endocarditis after extensive burns.



The secondary form occurs almost exclusively in the course of infectious diseases.

It is observed with great frequency in acute articular rheumatism. It may develop alone or be associated with pericarditis and myocarditis. But its frequency has often been overestimated, particularly by the French, who infer the existence of endocarditis from every systolic murmur occurring in the course of acute articular rheumatism, or even from irregular and accelerated heart's action. v. Bamberger states that the combination of acute articular rheumatism and endocarditis occurs in about twenty per cent of the cases, and his opinion is deserving of the greatest confidence. But as local and individual factors exert some influence, it is not astonishing that the proportions given by careful observers differ somewhat from each other. If it were true, as has been recently maintained, that the endocarditis is always primary, and gives rise to the articular affection, as a matter of course, articular rheumatism without endocarditis would be inconceivable. But this view is undoubtedly erroneous.

Endocarditis is more apt to complicate rheumatism the larger the number of joints affected and the more violent the inflammation. The age of the patient also exerts an influence, for, although children are not affected with rheumatism so frequently as adults, endocarditis is a frequent complication in them. The first endocarditic symptoms generally appear from the sixth to the fourteenth day of the disease. In very rare cases the endocarditis precedes the rheumatism. With regard to the connection between the two diseases we may assume, perhaps, that the bacteria which give rise to acute articular rheumatism adhere readily to the endocardium, and there produce inflammation. Perhaps previous changes in the endocardium (thickening and fatty degeneration) furnish a favorable soil for the deposit and development of the schizomycetes.

Chronic affections of the joints give rise much less frequently to endocarditis; a certain influence cannot be denied even to gouty changes in the joints.

This is also true of chronic muscular rheumatism. Specialists in diseases of children have often called attention to the coincidence of acute endocarditis and *caput obstipum rheumaticum*.

In using the term *endocardite rhumatismale d'emblée*, French authors imply that a certain morbid agent leaves the joints intact and attacks the endocardium primarily (a very bold hypothesis).

Endocarditis *verrucosa* also occurs, though not often, as a complication of typhoid fever. It is still rarer in typhus and relapsing fever. From a few reports it would seem as if endocarditis is especially frequent in relapsing fever when it attacks children.

The disease results occasionally from syphilis, but this relation has been overestimated by older writers, who inferred the syphilitic origin of endocarditic vegetations from their similarity to condylomatous proliferations.

Verrucose endocarditis has also been observed a number of times during gonorrhœa. In the majority of such cases the endocarditis is preceded by acute gonorrhœal inflammation of the joints, but the endocarditis may also develop irrespective of the latter affection. Among ten cases collected by Marly, in two the endocarditis existed alone, and in eight it was preceded by inflammation of the joints. Morel states that the endocarditis generally develops in the fourth or fifth week of the gonorrhœa, and most frequently attacks the aortic orifice. Our knowl-

edge concerning the connection between these two diseases is greatly advanced by Neisser's statements that gonorrhœa is the result of the action of bacteria (gonococci).

According to some observations, other infectious diseases may give rise to verrucose endocarditis, but our experience is too limited to warrant a positive opinion. French authors in particular have referred to the noxious influence of malarial infection. Isham states that he observed endocarditis during the course of parotitis. Its occurrence in ulcerative pulmonary phthisis and necrotic carcinoma has also been referred to.

But there is no doubt that endocarditis is not a very rare complication of acute infectious exanthemata, particularly of scarlatina. Of course this occurs especially in childhood, but it seems as if certain epidemics have a special tendency to this complication. The connection may be direct or indirect. In the first event the endocarditis develops directly during the course of scarlatina; in the latter event it is preceded by acute inflammation of the joints. In the former case the endocarditis may develop at the height of the eruption, or as a sequel. Moulinier estimates the frequency of endocarditis in scarlatina at ten per cent of all cases.

Variola is next in importance to scarlatina in this respect. The endocarditis develops usually towards the end of the eruptive period, or the beginning of the suppurative stage. It has been observed with relative frequency in hemorrhagic small-pox, probably because this form is a specially severe infection. It is met with very rarely in varioloid.

Endocarditis develops less frequently in erysipelas than in variola. Among all the acute exanthemata, it is observed least frequently in morbilli, and it would seem as if it may be looked for in this disease particularly when adults are attacked.

Verrucose endocarditis develops more frequently in men than in women, and the preponderance of males is manifested even in childhood. This results, in the main, from the fact that acute articular rheumatism plays such a prominent part in the etiology, and this is much more frequent in men than in women.

The disease is most frequent from the ages of twenty to thirty years.

II. ANATOMICAL CHANGES.—Verrucose endocarditis is characterized by peculiar proliferations and excrescences upon the inflamed portion of the endocardium. Their size varies from that of a poppy-seed to that of a pea, or even more, and their number also varies considerably.

In some cases the lines of closure of the valves are thickly strewn with the finest excrescences, in others there are a few larger proliferations. The function of the valves may suffer in both cases, either because complete closure is prevented, or because the excrescences narrow the valvular orifice. On account of the close proximity of small excrescences, or of the nodular and fissured surface of larger vegetations, the inflammatory proliferations assume a peculiar shape which has been compared to that of condylomata, cauliflower, or raspberry, a cock's comb, etc. Sometimes we find pedunculated, polypoid excrescences projecting from a valve or the wall of the heart into the cardiac cavities (endocarditis polyposa of some writers).

Recent, small vegetations are characterized usually by their gray and transparent appearance, sometimes by a soft, almost gelatinous consistency. Older vegetations become firmer and more rigid, at the same time white like tendon. They then consist of two parts; the lower part, which forms the base to a certain extent, is an inflammatory proliferation of

the endocardial tissue, while the upper part consists of thrombotic deposits from the blood. The boundary between both parts is easily recognized under the microscope; separation with forceps is possible only in recent cases.

In the new-born and in children we must be on our guard against mistaking the so-called Albin's nodules for endocarditic vegetations. The former are physiological and unimportant. They are found at the free edge of the auriculo-ventricular valves, in the shape of six to ten gray nodules, as large as a grain of sago. They consist of spindle cells and elastic fibres, and are the remains of the fetal development of the valves. v. Luschka and Reuss have shown that these nodules often contain extravasations of blood, sometimes of a red color, sometimes brownish red or blackish, from changes in the coloring matter. Elsaesser found such changes in more than half the autopsies on the new-born.

The development of endocarditic vegetations is the acme of the inflammatory process, and it now remains for us to trace its histological beginnings.

It has been maintained that the inflammation of the endocardium begins with hyperæmia, rather from analogy with other organs than from positive demonstration, although dilatation of the vessels has been observed beneath the vegetations. At all events, we must not mistake for inflammatory redness the imbibition redness which is observed in corpses that have been kept for a long time in a warm room, or in individuals who have died of severe general infection. The diffuse, extensive, and uniform redness of the latter will prevent mistakes.

The first demonstrable changes in the tissue of the endocardium are of a parenchymatous character. The interstitial tissue swells and becomes almost gelatinous; the connective tissue cells are also swollen. The inflamed part is therefore raised above the level of the surrounding tissues, thus forming the beginnings of the endocarditic vegetation. These parts usually strike us by their opacity and the dull lustre of the surface, the result, in part, of the desquamation and loosening of the endothelium.

Increase of the connective-tissue cells and emigration of white blood-globules from adjacent vessels follow very rapidly. The inflamed tissue thus becomes richer in cells than normal, and the growth of the endocarditic excrescences is stimulated. The young cells, as a rule, are especially abundant on the free surface of the endocardium.

The integrity of the covering endothelium cells suffers in consequence of these changes, and we find swelling, cloudiness, fatty degeneration, and desquamation of individual cells, in certain places processes of proliferation and fission. Now, since Bruecke's experiments, we know that intactness of the endothelium cells exerts a great influence on the fluidity of the blood. Zahn has also proven that changes in the endothelium of the vessels lead to the formation of thrombi at the affected parts, and it is not surprising, therefore, that firm deposits from the blood settle upon the inflamed portions of the endocardium.

In certain cases, parts of the vegetations break off, are carried into the circulation, and remain as an embolus in a peripheral artery. Emboli are found most frequently in the kidneys, spleen, brain, and arteries of the extremities. These processes are purely mechanical, and the emboli possess none of the infectious properties of septic endocarditis. They give rise to hemorrhagic or anæmic infarctions, but not to suppuration.

In almost every case acute endocarditis passes into the chronic stage. The vegetations become organized, grow firmer and more rigid, and form



hard thickenings and excrescences on the valves, with a great tendency to retraction. The valvular lesion, which has been produced acutely, then persists for the remainder of life. Calcification and fatty degeneration may also develop at the affected spots. These chronic sequelæ present an extreme tendency to pass into a condition of acute inflammation as the result of trifling exciting causes.

Complete absorption of the vegetations, or at least, entire restoration of the function of the valves, does take place, but is so extremely rare that its possibility is denied by some. I will mention two cases in my own experience. An officer was rejected by a life assurance company because he had mild articular rheumatism a year before, and, at the time of his application, had a loud diastolic murmur over the aorta, with hypertrophy and dilatation of the left heart. A year later I again examined this gentleman, and, to my surprise, the previous signs had disappeared. At a third examination, two years later, the heart was again found healthy. In a second case, in a girl of 14 years, signs of mitral stenosis developed, under my observation, during an attack of acute articular rheumatism, and for two years the patient was used by me as an excellent example of this lesion. Suddenly, the murmur disappeared, the subjective symptoms gradually diminished, and, although the patient remained under observation a year longer, no valvular changes could be recognized.

Absorption processes in the vegetations are sometimes manifested by a change in the character of the functional disturbance of the affected valves. For example, stenosis of an arterial or venous orifice may change gradually to insufficiency, when the vegetations have disappeared to such an extent that they offer no obstacle to the current of blood, but nevertheless prevent the closure of the valves by interfering with the unfolding of the leaflets.

Verrucose endocarditis almost always develops in the left heart. Inflammation in the right heart has been described, but is an exceptional event. During foetal life, however, inflammatory changes in the endocardium are found more frequently on the right side than on the left. The lines of closure of the valves are the sites of preference of the inflammation, and the vegetations are directed usually against the current of blood. In children the vegetations are present not infrequently at the opening of persistent foetal channels. Endocarditis is most frequent upon the mitral valves, particularly the anterior leaflet. The various parts of the heart are affected in the following order of frequency: Mitral valves, aortic valves, tricuspid valves (much more rarely), pulmonary valves, walls of the heart, and trabeculæ. Parietal endocarditis presents the following order of frequency: Left auricle, right auricle, left ventricle, right ventricle. In parietal endocarditis the inflammation may extend to the myocardium, and give rise to acute ulceration and aneurism of the heart.

III. SYMPTOMS.—A diagnosis is possible if the disease leads to disturbance of function of the valvular apparatus. If the vegetations do not interfere with the mechanics of the valvular structures—as happens in small vegetations of the valves, and particularly in parietal endocarditis—even a probable diagnosis is impossible.

A diagnosis can be reached only by physical exploration. Subjective symptoms may be entirely absent or so indefinite as to possess no special diagnostic significance. During the course of acute articular rheumatism in particular, the heart should be carefully examined every day.

Some patients complain of a peculiar dull feeling of oppression and

tension in the cardiac region, but this is rarely intensified into severe pain. Dyspnoea is sometimes noticed, either permanently or in paroxysms. In the latter event it is associated not infrequently with attacks of palpitation, which may be felt as a subjective sensation or demonstrable objectively. The palpitation may also be associated with a constricting pain in the region of the heart, radiating to the epigastrium and umbilicus, to the left arm and to the back of the neck.

The bodily temperature is not infrequently elevated; the pulse becomes more frequent and may be irregular.

The first cardiac symptom is the development of a systolic murmur which is heard with greatest intensity at the apex. If this is associated with dilatation of the right ventricle, no conclusion is warrantable, since both conditions are observed not infrequently in anæmic and febrile affections. The diagnosis can be made with certainty only when hypertrophy of the right ventricle is added as a third symptom, in other words when the pulmonary diastolic sound is permanently intensified and assumes a clicking character. The signs of mitral insufficiency, therefore, have developed under our observation.

As a matter of course, the symptoms are different if other valves are affected or if stenosis of the orifices is produced. The gradual development of diastolic murmurs is specially important, as they are rarely accidental, and in themselves, therefore, allow us to diagnose endocarditis almost with certainty.

Pericarditis and endocarditis often develop at the same time or in rapid succession, and the diagnosis of endocarditis is thereby rendered much more difficult. Extensive exudations and very loud pericarditic murmurs may entirely conceal the endocarditic murmur, and not infrequently the signs of endocarditis become so much more distinct the more the symptoms of pericarditis disappear.

If endocarditis gives rise to embolism, the diagnosis is not difficult in many cases. Large emboli in the kidneys are recognized by sudden pain in the renal region and by hæmaturia, sudden chill and frequent vomiting. In splenic embolism, pains are felt in the region of this organ, and acute splenic enlargement is generally demonstrable. Emboli in the arteries of the limbs give rise to pulselessness below the obstructed spot, a sensation of coldness and diminished temperature in the limb, a feeling of stiffness, often lancinating pains and paresis. Cerebral emboli are followed by sudden disturbance of consciousness and hemiplegia.

Acute endocarditis is associated occasionally with peculiar complications; we refer particularly to erythema nodosum and chorea. Both affections have been regarded as the result of embolism, the former of cutaneous vessels, the latter of those in certain parts of the brain (vide Vol. III.).

Endocarditis verrucosa gives rise in almost all cases to valvular lesions. It then passes into a chronic stage which lasts for life.

IV. DIAGNOSIS.—The diagnosis is readily made in cases of valvular endocarditis when a valvular lesion develops under observation. If this is not the case, judgment must be reserved. Systolic murmurs, cardiac arrhythmia, and accelerated heart's action are, in themselves, insufficient for purposes of diagnosis.

V. PROGNOSIS.—This is unfavorable as regards recovery, as we are unable to prevent the inflammation or to relieve it when existing. Furthermore, the prognosis coincides with that of valvular lesions (vide next section).

The prognosis must be given very cautiously in another respect. It may happen that infectious diseases run an apparently favorable course, and that signs of endocarditis and valvular lesion do not appear for months. This may be explained on the theory that the originally acute and insignificant endocarditis gradually led to such considerable retraction of the valves that a valvular lesion only developed secondarily.

VI. TREATMENT.—Prophylactic measures are useless (salicylic acid or salicylate of sodium).

If we have reason to suspect the existence of endocarditis, an ice-bag should be applied constantly to the cardiac region. Quinine and salicylic acid preparations are indicated in high fever. If the action of the heart is very rapid, digitalis should be carefully employed. After the signs of valvular lesion have become permanent, the attempt may be made (though with little hope of success) of causing absorption of the vegetations by the use of iodide of potassium (10 : 200, one tablespoonful t. i. d. after meals), and iodide of iron (syr. ferri iod., syr. simpl., āā 20.0. M. D. S. One teaspoonful t. i. d., or ferri iodat. sacch., sacch. alb., āā 0.5. One powder t. i. d.). The treatment of valvular lesions must then be relied upon (vide next section).

Gerhardt has had remarkable results from inhalations of carbonate of sodium. He states that 2–4 inhalations of a 1–1½% solution have effected considerable improvement in a number of cases and in three cases even complete recovery.

### *C. Chronic Endocarditis. Endocarditis Chronica Retrahens.*

I. ETIOLOGY.—Chronic endocarditis develops in very many cases from a preceding acute attack, and its etiology is then the same as that of the latter form. In other cases it begins in an insidious and independent manner, and is dependent on special etiological factors. Not infrequently we have to deal with senile changes, the endocardium undergoing the same changes as the intima of the vessels in the arteriosclerosis of old age. Immoderate and prolonged bodily exertion also appears to exert some influence, at least English and American military surgeons have very often observed valvular lesions as the result of chronic endocarditis among soldiers, particularly recruits.

Chronic endocarditis very often follows nephritis. Among 292 cases of Bright's disease, Frerichs found cardiac hypertrophy 99 times, 41 of which (14 per cent) were the result of valvular lesion (generally mitral stenosis or insufficiency). We are probably warranted in explaining these changes by the retention in the blood of certain constituents of the urine, which exert an irritant effect on the endocardium.

Certain authors maintain that chronic endocarditis is associated with pulmonary emphysema, carcinoma, gout, arthritis deformans, and serofula. French writers believe that chronic lead poisoning gives rise to the disease. The noxious influence of alcohol on the endocardium seems more assured, so that chronic endocarditis should be included among the diseases of drunkards. Lecorché mentions its relative frequency in diabetes mellitus, and explains this by the irritant effect of the sugar in the blood.

II. ANATOMICAL CHANGES.—The inflamed parts of the endocardium present a tendinous or cartilaginous thickening and consistence; they look cloudy and opaque (fibrous or sclerotic endocarditis). When the valvular apparatus is affected, the mobility and therefore the function



of the valves necessarily suffer. These disturbances increase in intensity if calcification and even ossification are added to the simple hyperplastic changes. The disturbance of function is still further increased by the extreme tendency of the inflamed tissue to retraction (*endocarditis chronica retrahens*). Fatty degeneration of the diseased parts is less significant, though it gives rise occasionally to perforation, as it does in *atheroma* of the arteries.

If the endocarditis affects the semilunar valves, the latter often project into the orifice as rigid, almost immobile walls. During systole they present an abnormal resistance to the current of blood, because they can be pushed very little or not at all to the side, and thus cause stenosis at the beginning of the arterial system. As their unfolding is also interfered with, it becomes possible during ventricular diastole that the blood may regurgitate from the arteries into the left ventricle through the opening left between the free borders of the valve, *i.e.*, there is insufficiency of the valve. Clinically, however, one or the other form of valvular lesion predominates, so that the clinical symptoms do not always correspond to the anatomical changes. Not infrequently there are adhesions between two or even all three leaflets. The partition disappears and the sinuses of Valsalva merge into one. These changes are associated generally with marked stenosis.

Upon the auriculo-ventricular valves the disturbances become especially marked, because the thickening and retraction are not confined to the tissue of the valve, but extend to the chordæ tendineæ. The latter are converted into short, rigid, thick strands. Retraction and other changes, attributable to fibrous myocarditic processes, occur in the corresponding papillary muscles, converting them into short, plump, fibrous columns. The stenosis of the auriculo-ventricular orifices may become so great that only a narrow, button-hole slit is left for the current of blood. Adhesions of the free borders of the valves occur sometimes, and, in association with thickening of these places, produce still further stenosis.

Circumscribed chronic inflammation of the valves, which is not very extensive, may be compensated by considerable dilatation of the adjacent healthy valvular tissue. This has been observed at the semilunar and auriculo-ventricular valves. Gowers recently reported a case in which one leaflet of the mitral valve was excessively retracted, while the other was remarkably distended and elongated, though not to a sufficient extent to assume entirely the function of the retracted leaflet.

The remarks concerning the site of verrucose endocarditis also hold good with regard to chronic endocarditis. The mitral valve is affected most frequently, next the aortic valve, most rarely the tricuspid and pulmonary valves. Chronic parietal endocarditis is not so frequent as the valvular form, and has no clinical significance. The inflammatory thickening of the endocardium often extends to the myocardium (*chronic endo-myocarditis*). Lepine states that he has observed chronic endocarditis most constantly on the posterior external wall of the left auricle, and explains it by the mechanical factors which obtain in the circulation of the blood. In that form which results from senile changes and arteriosclerotic processes, the affection is more frequent at the aortic than the mitral valves. The process then spreads directly, to a certain extent, from the intima of the aorta to the aortic valves. Mitral lesions are more frequent, accordingly, from the 15th to 30th years of life, and aortic lesions in advanced age. We have referred previously to the great

tendency of the disease to acute relapses (endocarditis retrahens recurrens).

Changes in the heart muscle are almost always present. Dilatation and hypertrophy must be attributed to the valvular lesions. Very often there is also fibrous degeneration which may lead to chronic aneurism of the heart. Rindfleisch even attributes all chronic fibrous inflammations of the heart muscle to a preceding chronic endocarditis. If a part of the affected valve breaks off, opportunity is afforded for the production of emboli.

Sperling furnishes the following tables of the frequency with which the individual valves are affected and the occurrence of emboli :

#### 300 Cases of Endocarditis.

268 times (89%), . . . . .	left heart alone affected.
3 " (1%), . . . . .	right " " "
29 " (10%), . . . . .	both sides affected.

#### Affections of one Valve alone : 200 Cases (66.7%)

Mitral valve alone, . . . . .	157 times (77.5%)
Aortic " " . . . . .	40 " (20.0%)
Tricuspid valve alone, . . . . .	3 " (1.5%)
Pulmonary valve alone, . . . . .	0 " (0%)

Total, . . . . . 200

#### Combined Affection of the Valves : 100 Cases (33%).

Mitral and aortic valves, . . . . .	71 times (71%)
Mitral and tricuspid valves, . . . . .	9 " (9%)
Mitral and pulmonary valves, . . . . .	2 " (2%)
Aortic and pulmonary valves, . . . . .	1 " (1%)
Aortic and tricuspid valves, . . . . .	0 " (0%)
Mitral, aortic, and tricuspid valves, . . . . .	16 " (16%)
Mitral, aortic, and pulmonary valves, . . . . .	0 " (0%)
Tricuspid, pulmonary, and mitral valves, . . . . .	0 " (0%)
Tricuspid, pulmonary, and aortic valves, . . . . .	0 " (0%)
All four valves, . . . . .	1 " (1%)

Emboli in 84 Cases (28%); 76 from the Left, 6 from the Right Heart.

Renal emboli, . . . . .	57 times.
Splenic emboli, . . . . .	39 "
Cerebral emboli, . . . . .	15 "
Hepatic and intestinal emboli, . . . . .	5 "
Cutaneous emboli, . . . . .	4 "

It must not be inferred from the above tables that certain combinations of valvular lesions never occur—they simply did not come under observation during the years included in the above statistics.

Chronic endocarditis can only be diagnosed when it leads to disordered function of the valves. Its symptoms, diagnosis, prognosis, and treatment are then the same as those of valvular lesions (vide next section), but it has seemed to us proper to discuss them separately, because the latter affection is sometimes, though much more rarely, due to other causes.

## 2. *Acquired Diseases of the Valves.*

I. ETIOLOGY.—The term valvular lesion is applied to every functional disturbance of the valves which is clinically demonstrable ; it embraces insufficiency and stenosis of the valves.

As a rule, these disturbances are the result of endocarditis.

Sometimes, however, valvular lesions result from severe bodily exertion. The semilunar valves are torn from their points of insertion, or

the auriculo-ventricular valves from their chordæ tendinæ, and the valvular apparatus then becomes suddenly insufficient. This has been observed after lifting a heavy load, long running, etc.

Peter describes a case in which a man was attacked by a savage horse and, in a desperate attempt to escape, the aortic valves were torn. A number of cases have been reported in which a similar accident occurred to individuals who struggled desperately to save themselves from falling a great distance.

The aortic valves are torn most frequently, next the mitral and tricuspid valves; I am acquainted with no illustration of rupture of the pulmonary valves. Peacock and Barié furnish the following tables:

Peacock, . . . . . 17 cases.	{	Aortic valve,	10 times	(58.8%)
		Mitral "	4 "	(23.5%)
		Tricuspid valve,	3 "	(17.7%)
Barié, . . . . . 35 cases.	{	Aortic valve,	16 times	(45.7%)
		Mitral "	16 "	(45.7%)
		Tricuspid valve,	3 "	(8.6%)

Sudden rupture of the valves will occur so much more readily if it has been preceded by endocarditis, but intact valves may also yield to an abnormally high blood pressure. The clinical symptoms are the same as those of valvular lesions which have developed gradually from endocarditis, but, as a matter of course, the onset of the affection is different. The patients generally feel that something has torn within the chest or heart. Sometimes they fall unconscious, or suffer, at least, from violent oppression and dyspnoea; there are usually very severe pains in the cardiac region. A cardiac murmur develops suddenly, and is followed, after days or weeks, by signs of dilatation or hypertrophy of certain portions of the heart. If compensation remains absent for any reason, or is incomplete, symptoms of stasis soon develop, and the patient succumbs. In the other event, the patient may live for years. According to Anstie, recovery occurs in exceptional cases.

The function of the valves is disturbed occasionally by new growths. v. Bamberger described a case of mitral stenosis, the result of a myxoma which grew from the left auricle into the mitral orifice. A similar condition has been observed in aneurism of the heart valves and in cardiac thrombosis.

Relative insufficiency of the valves merits special mention. This term refers to a condition in which the tissue of the valves is or may be unimpaired, but they are unable to close the orifice completely on account of the distention of the latter.

Relative insufficiency is most frequent at the tricuspid orifice. It develops there when the blood pressure within the pulmonary artery is excessive, as in chronic diseases of the lungs, and particularly in mitral lesions. It has been observed at the aortic orifice as the result of acute inflammation of the walls of the aorta. Thus Moxon reports a case in which inflammation of the aorta gave rise to such extensive relaxation of the wall of the vessel and distention of its lumen that the aortic valves were unable to close the orifice. A number of cases of pulmonary insufficiency have been reported as the result of sudden occlusion of the trunk of the pulmonary artery or one of its main branches, by echinococcus vesicles or other emboli, and subsequent dilatation of the artery on this side of the obstructed point. Litten justly lays stress on this feature in the diagnosis of embolism of the pulmonary artery.

Some writers maintain that excessive bodily exertion may give rise to relative insufficiency of the valves, and interpret in this manner the valvular lesions which develop not infrequently in recruits. But we



must call attention to the fact that such exertions also predispose to arterio-sclerotic changes, and that these may give rise to valvular lesions.

Perls noticed that the aortic valves have a predisposition of age, as it were, to relative insufficiency. At an advanced age the relation between the aortic orifice and the space which the valves are capable of covering changes in favor of the former. As a matter of fact, such relative insufficiency develops very exceptionally, but at all events it is readily understood that, under certain circumstances, very slight changes and retractions in the aortic valves are capable of giving rise to insufficiency.

Relative insufficiency of the heart valves necessarily coincides in its mechanical effects with all other etiological forms of valvular insufficiency. Dilatation and hypertrophy of certain portions of the heart are the necessary consequences. Many physicians, particularly the French, are very prone to assume relative valvular insufficiency. They not infrequently interpret in this sense the systolic murmurs which are apt to develop in febrile and anæmic conditions. But as dilatation and hypertrophy of the heart remain absent, we are not justified in assuming relative insufficiency under such circumstances.

v. Bamberger has called attention recently to functional insufficiency of the valves, in which the tissue of the valves and the orifices are unchanged, but closure of the orifice is prevented by fatty changes in the papillary muscles. According to older writers, disordered innervation of the papillary muscles may act in the same way.

Neukirch gave a description very recently of relative stenosis of the orifices. He believes that the orifices and valves were unchanged, but that the cavities of the heart had undergone such marked dilatation, and contained such a large amount of blood, that the orifices were relatively narrowed (?).

It is evident from the above description that valvular lesions do not constitute an anatomical unity. In the large majority of cases, indeed, we have to deal with the sequelæ of endocarditis, so that it is not astonishing that the valves of the left side are usually affected. The mitral valves are affected most frequently, next the aortic, then the tricuspid or the pulmonary valves.

In many cases only a single valve is affected, in others there are combined valvular lesions. The combination of mitral and aortic disease is observed most frequently, for, on account of the close proximity of these valves, the inflammation spreads not infrequently from one to the other. There is also a frequent combination of mitral disease with relative tricuspid insufficiency. Furthermore, Friedreich has shown that the association of aortic insufficiency and relative tricuspid insufficiency is not rare.

Vocation, age, and sex exercise some effect on the development and localization of valvular lesions. The latter are found most frequently among the working classes. While young persons (fifteen to thirty years) often suffer from mitral lesions in consequence of endocarditis, aortic insufficiency from arterio-sclerotic changes develops often between the fortieth and sixtieth years. Women suffer more frequently than men from valvular lesions.

Mitral lesions are more frequent in females, aortic lesions in males. Some writers maintain the heredity of valvular lesions, and I am acquainted with a number of families in which several generations

have suffered from heart disease, but in whom I was not always able to demonstrate the ordinary causes.

II. SYMPTOMS — The symptoms of valvular lesions of the heart may be divided into local and general. The former include all morbid phenomena on the part of the heart and circulatory apparatus in general. They are the direct result of the valvular lesion, and are important, inasmuch as they alone permit the positive recognition of the valvular lesion. The general symptoms include the affections of other organs which are secondary to the disturbed circulatory conditions.

The direct effects of a valvular lesion are manifested by dilatation and hypertrophy of certain portions of the heart. These result from the increased resistance to circulation in certain parts of the organ, and, in valvular insufficiency, from the fact that certain parts of the heart contain a larger amount of blood than normal. It is on account of these changes in the heart muscle alone that the circulatory disturbances can, to a certain extent, be compensated. If compensation does not develop or is insufficient, or retrogresses for any cause after it has once once developed, disturbances of compensation arise which are manifested chiefly by stasis in the outflow of venous blood.

All valvular lesions coincide with regard to the circulatory conditions. They give rise to retardation of the arterial current, diminished tension in the aortic system, and increase of the medium pressure in the domain of the *venæ cavæ*.

In stenosis of the orifices, these changes can be deduced directly, because the lesion necessarily diminishes the blood pressure, and retards the rapidity of the circulation peripherally, while it gives rise to stasis behind it, and this, according to the character of the valvular lesion, will be propagated directly or indirectly to the *venæ cavæ*. But a little consideration will show that the same physical changes will develop in valvular insufficiency as a result of regurgitation of blood. As a matter of course, a fatal termination would rapidly ensue if the compensatory processes did not attempt to relieve the circulatory changes.

### *a. Aortic Insufficiency.*

If the aortic valves are insufficient it becomes possible, during diastole of the left ventricle, for the blood to regurgitate from the aorta into the left ventricle. This regurgitation is the result partly of gravitation, partly of the contraction of the aorta. As soon as the blood has passed from the narrow aorta into the wide diastolic left ventricle, it undergoes a rotary movement which is manifested acoustically as a diastolic murmur.

The rotary movement is increased from the fact that two currents of blood flowing in opposite directions (the regurgitated blood and that flowing from the left auricle into the ventricle) strike against one another.

At each diastole the left ventricle is forced to receive more blood than under normal conditions, as the regurgitated blood is added to that which flows in from the auricle. It must therefore increase in dimensions, *i. e.*, become dilated. Except under special circumstances, the degree of dilatation of the left ventricle corresponds to a certain extent to the amount of insufficiency.

In order that the circulation should not be entirely disarranged, the left ventricle at each systole must propel more blood than normal into

the aorta, since it contains the regurgitated blood in addition to the normal amount. This increased work can only be effected by increase in the mass of muscular tissue (hypertrophy).

To understand the clinical phenomena in the peripheral arteries, it must be remembered that the entire arterial system receives an abnormal amount of blood during each systole of the left ventricle. The arterial blood is discharged with unusual rapidity, inasmuch as it flows, during diastole, not alone towards the periphery of the body, but in part into the left ventricle by regurgitation. The following are the phenomena observed in the various methods of examination.

**INSPECTION.**—As a rule, there is marked prominence of the præcordial region, on account of the increased size of the left ventricle. This is particularly marked in women and children on account of the slighter resistance of the thorax. It often extends beyond the cardiac region proper to the left axillary region, and can be readily measured (it must be remembered, however, that the right thorax is normally 2.5 cm. greater than the left).

In addition, vigorous diffuse pulsating movements are noticeable in the region of the heart. If the median border of the left lung is pushed far to the outside by the hypertrophic left ventricle, if the thoracic integument is poor in fat, the intercostal spaces broad and the muscles thin, the cardiac movements may be visible in several intercostal spaces and lead to systolic retractions—insignificant in themselves—along the left border of the sternum.

The pulsations conveyed to the thorax may be recognized not infrequently through the clothes, so that an experienced observer is at once led to suspect aortic insufficiency. Sometimes the systolic movements are conveyed to the bed.

The apex beat presents very noteworthy changes. It extends almost always beyond the left mammary line on the outside, and occasionally reaches the left axillary line. At the same time it is unusually broad, and may occupy the sixth, seventh, even the eighth intercostal space. These changes must be attributed to dilatation of the left ventricle.

The apex beat is usually more prominent and lifting than normal, and on palpation it is found to have unusual strength and power of resistance. This is owing to the hypertrophy of the left ventricle.

Vigorous pulsations are noticed occasionally immediately adjacent to the right edge of the sternum in the second right intercostal space, and are generally attributable to dilatation of the ascending aorta.

Unusually vigorous pulsation of the carotids is observed generally on inspection of the neck. The pulsatile movement is conveyed to the head and occasionally the impression is created as if the entire trunk shook at each systole.

Pulsating elevations are sometimes observed in the jugular fossa, in consequence of excessive tension of the aorta and aortic arch. The pulsating vessel can often be reached if the finger is pressed in deeply. Visible pulsations are also found in the smaller arteries (temporals, coronary arteries of the lips, arteries of the fingers, etc.). These result from the unusually large amount of blood in the arteries and the increased blood pressure as the result of hypertrophy of the left ventricle.

Quinke has also called attention to a capillary pulse; this is recognized on the nail-bed by the changed position of the boundary between the red and white occurring at each pulsation. But this must not be regarded as a pathognomonic sign of aortic insufficiency. The capillary



pulse is found occasionally in healthy individuals, particularly upon lifting the hand. Furthermore a practised eye is required, indeed suitable preliminary exercises of the eyes are necessary.

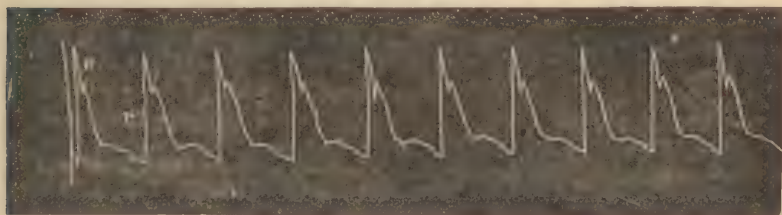
Sometimes the pulse is conveyed through the capillaries into the veins of the back of the hand (venous pulse), but this also is sometimes observed in healthy individuals.

Quinke also noticed pulsation of the retinal arteries in many cases of aortic insufficiency. The more marked and uncomplicated the insufficiency is, the more this phenomenon may be expected. But it does not occur in all cases of aortic insufficiency, and is found sometimes in other affections, for example, Basedow's disease. In many cases the ophthalmoscopic examination with the direct image is requisite; with the inverted image the pulsation will be recognized only when it is very marked.

The phenomenon is manifested by the fact that the red columns of blood in the retinal arteries are interrupted rhythmically and coincidentally with the radial pulse, or still more frequently the arteries become dilated and sinuous at each cardiac systole, while the retinal veins present emptying and filling in the opposite rhythm. If the phenomenon is not spontaneous, it may be produced by slight pressure on the eyeball. The pulsation is noticed most readily at the boundary of the optic papilla. Sometimes it is found only in one eye or one arterial branch.

O. Rosenbach reports two cases of arterial hepatic pulsation, which

FIG. 12.



Pulse curve of right radial artery in insufficiency of the aortic valves. *e*, elevation of elasticity; *r*, elevation of recoil.

he attributes to abnormal fulness of the hepatic artery and its fine branches within the parenchyma of the liver.

Guéneau de Mussy observed that in many cases patients with aortic insufficiency assume a horizontal position, while those suffering from mitral disease assume as erect a position as possible. He attributes this to the fact that in the erect position the regurgitation of the blood in aortic insufficiency is favored by gravitation.

**PALPATION.**—We have already referred to the increased resistance of the apex beat.

In some cases diastolic fremitus is felt over the base of the heart, but grows feebler towards the lower border of the heart and the apex. This corresponds to the diastolic murmur. However, it is often absent in aortic insufficiency, and sometimes occurs only when the heart's action is excited by physical or mental exertion. As a general thing, the fremitus is so much more apt to occur the louder the endocardial murmur, though in certain cases there is a striking disproportion between the fremitus and the intensity of a murmur in favor of the former.

Very characteristic changes are noticed in the radial pulse. It is unusually full and hard (*pulsus altus, celer, durus*). The line of ascent of the sphygmographic tracing is strikingly steep and high; the transition from the ascent to the descent is effected at a very acute angle. The line of descent (particularly its first portion) is unusually steep, and the elevation of recoil is not particularly well marked (vide Fig. 12). That the elevation of recoil is not entirely absent despite the aortic insufficiency is explained by Landois on the ground that during cardiac diastole the wave of blood is reflected from the ventricular wall situated opposite to the aortic orifice. As a matter of course, the sphygmographic tracing just described can only be expected if there are no complications on the part of the heart muscle, the walls of the arteries, or the other valves of the heart. For example, if the aortic insufficiency is the result of arterio-sclerotic changes, and these are also present in the peripheral arteries, the tracing changes at once, and the pulse assumes the form of the *pulsus tardus* in consequence of the diminished elasticity of the arteries. Older authors state that the radial pulse becomes fuller and more bounding when the arm is elevated. Some recent writers deny this, others state that they have observed this phenomenon under other conditions. According to my own experience, the phenomenon is present in rare cases, but occasionally I have also observed it very distinctly in anæmic and febrile conditions.

Palpation of the carotid not infrequently reveals a peculiar purring and vibration which is merely an arterial purring thrill, and corresponds acoustically to a vascular murmur coincident with cardiac systole. Pressure upon the vessel must be avoided, else artificial stenosis will be produced, giving rise to a whirl in the blood and the development of a murmur. Davidson states that there is often an inequality of the carotid pulse on the two sides.

Spontaneous systolic thrills occur occasionally in the abdominal aorta, and may sometimes be followed to the sacrum. All these arteries present the bounding pulse, but this characteristic is not infrequently more marked in the vessels of the upper half of the body than in those of the lower half.

The pulsation in the smaller arteries can usually be felt with remarkable distinctness, and in some cases it appears to be much later than the apex beat of the heart. Tripier observed this fourteen times in twenty-six cases, and explains it on the ground that the blood current, at the beginning of cardiac systole, must first overcome the resistance offered by that portion which has regurgitated from the aorta.

**PERCUSSION.**—This reveals enlargement of the left ventricle, *i. e.*, cardiac dulness extends on the outside beyond the left mammary line; it usually begins very high, not infrequently at the second left costal cartilage, and extends lower than normal (eighth or even ninth rib); the right border is unchanged. Botkin remarks that the area of dulness sometimes overlaps slightly the position of the apex beat.

In rare cases the right side of the heart is enlarged; the greater (relative) area of dulness extending on the right beyond the edge of the sternum and the cardiac resistance extending more than 2 cm. beyond the right edge of the sternum at the level of the fourth right costal cartilage. Vigorous lifting of the lower half of the sternum or a clicking second (diastolic) pulmonary sound also indicates hypertrophy of the right ventricle. This is sometimes found when disturbed compensation

and symptoms of stasis have never been present, and such cases are still obscure.

Dulness appears occasionally within the second right intercostal space and immediately adjacent to the right edge of the sternum; it is a few centimetres in width and depends on dilatation of the beginning of the aorta.

**AUSCULTATION.**—The most important sign in auscultation is the diastolic murmur in the cardiac region. It is not always loudest over the point of auscultation of the aorta, *i. e.*, the second right intercostal space, but is generally heard with greatest intensity over the middle of the sternum, immediately adjacent to the left edge of the sternum. This is owing to the fact that the whirl of blood and therefore the murmur does not form in the beginning of the aorta, but in the left ventricle. The murmur is usually blowing and deep, while sawing or whistling (musical) murmurs are rare.

The diastolic murmur is almost always audible over the origin of the pulmonary artery, and in a few cases in which the diagnosis was verified post mortem, it had the greatest intensity over the beginning of the pulmonary artery. It is absent or very feeble over the apex. Balfour says that when the murmur is propagated to the apex and perhaps is most intense there, the posterior aortic leaflet is insufficient, but this statement requires corroboration. The murmur is so loud at times as to be audible at a distance from the patient, and Burney Yeo reports a case in which it was heard at a distance of one metre. It often extends beyond the cardiac region, so that, for example, it is audible on the posterior thoracic surface.

If the insufficiency affects only one or two leaflets, while the others are healthy, a diastolic sound may be heard at the origin of the aorta in addition to the diastolic murmur. If the murmur is very loud, it often conceals the sound, and in such cases it is well to remove the ear a little from the stethoscope, whereupon the murmur disappears and the sound which, as is known, is propagated better than a murmur, makes its appearance. But the sound might also be propagated from the pulmonary artery to the origin of the aorta. It is therefore safer to practise auscultation of the carotid, for if a cardiac diastolic sound is heard there, it originates almost without question at the aortic valves.

The other heart sounds are often unchanged if no special complications are present. In certain cases the first sound is very feeble or absent at the apex, and Traube has employed this fact, though not without opposition, to prove that the systolic sound at the apex is predominantly a valvular sound under normal conditions. A systolic murmur is occasionally heard at the apex. II. Jakobson explains this on the theory that the excessively distended fibres of the hypertrophic left ventricle are no longer capable of periodic vibrations, and therefore of the production of tones. In some cases, however, the murmur is conveyed from the aorta. The first sound is more rarely intensified at the apex. As the second sound at the apex is conveyed from the aorta in healthy individuals, it might be supposed that it is absent in aortic insufficiency or replaced by a murmur, unless a diastolic sound is produced at the aortic valves in addition to the diastolic murmur. In reality, there are several possibilities which explain the presence of a diastolic sound at the apex. In the first place, it may be conveyed from the pulmonary artery, or it might arise, if the aortic insufficiency is very marked, from the impact of the regurgitated blood against the wall of the ventricle situated opposite to the aortic orifice.



Finally, Traube believed that, in consequence of the regurgitation, the mitral valves were closed during diastole and gave rise to a sound, thus explaining the absence of the systolic ventricular sound. The systolic aortic sound is very often converted into a systolic murmur, without justifying us in assuming aortic stenosis in addition to insufficiency. In all probability, this murmur depends on the excessive tension of the wall of the aorta during systole and irregular molecular movements produced thereby.

A systolic murmur is very often heard in the carotid. This may be propagated from the origin of the aorta into the carotid. But there are also autochthonous, cardiac systolic, carotid murmurs; they must be regarded as belonging to this class when no systolic murmur is present at the aortic orifice. Palpable fremitus over the carotid also favors the autochthonous character of the vessel murmur. It has been attributed to irregular vibrations of the vascular walls in consequence of their excessive tension, but Talma has recently interpreted them as hæmic murmurs. In many cases nothing whatever is heard over the carotid during diastole. In others we hear a diastolic murmur which is conveyed from the left ventricle. But occasionally a diastolic sound is heard which is also conveyed from the aortic orifice into the carotid, but also furnishes proof that one of the semilunar aortic leaflets is still capable of being unfolded.

Over the peripheral arteries is heard the so-called arterial sound. Upon applying the stethoscope gently and carefully, even to the smaller arteries, a short sound, beginning and ending abruptly, is audible. This arterial sound is often heard over the temporal and radial arteries, even over the arterial arch of the palm of the hand. Upon greater pressure, it is converted into a murmur of stenosis and, upon occlusion of the vessel by still further increase of pressure, we again hear a simple systolic, so-called "pressure sound." But these sounds are also heard in febrile and anæmic conditions.

Upon gentle contact of the stethoscope with the crural artery, we hear, in many cases, only a systolic vascular sound, the result, evidently, of tension of the wall of the vessel. If the pressure is increased, the sound changes, as it does under normal conditions, into a murmur and then finally into a simple pressure sound of the artery.

If the pressure is increased very gradually, we soon arrive at a point at which the murmur is reduplicated, though both parts are included in cardiac systole. There are transitions between marked reduplication and the simple murmur, the first and second parts of the murmur being connected by a very gentle, continuous interval.

Reduplication of the systolic vessel murmur must not be mistaken for Duroziez' phenomenon. In the latter, a certain degree of pressure with the stethoscope gives rise to a systolic and diastolic murmur. This phenomenon can also be produced by compressing the vessel with the finger at a little distance from the site of auscultation. The following explanation is offered by Duroziez: The vessel is narrowed in consequence of the compression; hence a systolic murmur is produced on the peripheral side, because the blood, after overcoming the stenosis, undergoes a whirling motion. On account of the abnormal filling of the arterial system, and especially on account of the regurgitation of blood, a murmur arises centrally during diastole, because the blood which has regurgitated from the periphery, after it has passed the stenosed portion of the vessel, undergoes an irregular movement. Duroziez' phenomenon is also observed in lead cachexia, and in anæmic and febrile conditions.

In Traube's phenomenon we have to deal with spontaneous sounds which arise independently of pressure with the stethoscope. It is characterized by a sound which is heard during the systole and diastole of the artery. Traube maintained that the sound arose during cardiac systole from the sudden tension of the wall

of the artery, while it was produced during diastole by the sudden and excessive loss of tension.

Traube believed that the double sound was heard only in very marked aortic insufficiency, but Fraentzel showed that it may also be present in slight insufficiency. In all cases, however, the following conditions, as pointed out by Riegel, must be present: extensive cardiac hypertrophy, normal elasticity of the arterial walls, absence of fatty heart, and atheromatous changes in the arteries.

Friedreich has cautioned against mistaking this phenomenon for sounds in the crural vein and mixed sounds in the crural artery and vein. The latter can be suspected only when tricuspid insufficiency is also present, since venous sounds may develop under such circumstances.

The reduplicated sound and murmur occur not alone in the crural, but also in the subclavian and axillary artery. They are neither constant nor frequent, and have also been found in other conditions. We come to the conclusion, therefore, that no acoustic phenomenon in the peripheral vascular system is characteristic of aortic insufficiency.

The signs of aortic insufficiency sometimes disappear during the course of prolonged observation, and clinical recovery ensues. This may happen when only one or two leaflets are affected, and the third is distended to such an extent as to assume the function of the others, or when the endocarditic vegetations increase more and more in size and thus render the closure of the valves possible.

In some cases the signs of aortic insufficiency gradually merge into those of stenosis. This is also the result of increase of the vegetations, which render the valves sufficient, but at the same time narrow the origin of the aorta.

### *b. Stenosis of the Aortic Orifice.*

In aortic stenosis, the blood meets with an abnormal resistance at the origin of the aorta during left ventricular systole. In order to overcome this resistance, the left ventricle must dilate and hypertrophy. Both conditions are less marked than in aortic insufficiency, in which the left ventricle contains more blood than normal. Insufficiency and stenosis are usually associated with one another.

On account of the stenosis, the blood expelled from the left ventricle during systole undergoes a whirling motion, as soon as it has passed the site of stenosis and has entered the wide origin of the aorta. This is manifested acoustically by a systolic murmur.

Within a given time, less blood flows into the aorta from the left ventricle than under normal conditions, although the hypertrophy endeavors to compensate for this disproportion. Hence the systole occupies a longer period than normal.

**INSPECTION.**—The apex beat is entirely absent in many cases, because, on account of the stenosis, the recoil of the heart—a prominent factor in the production of the apex beat—is weakened. In other cases the apex beat is resisting and heaving, as is the rule in hypertrophy of the left ventricle. It is also pushed downwards and to the outside, but these changes are not so marked as in extensive dilatation of the left ventricle.

In one case, Friedreich observed systolic retraction of the apex, and attributes it to deficient distention of the arch of the aorta and consequent diminished systolic dislocation of the heart downwards and to the outside.

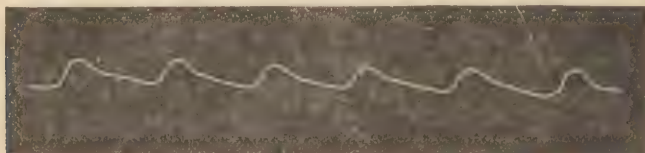
Protrusion of the præcordial region is usually present in individuals with a yielding thorax.

**PALPATION.**—In very many cases, a systolic purring thrill is felt, most marked at the base, particularly in the second right intercostal

space, but sometimes extending to the apex and even beyond the precordial region proper.

The radial pulse is usually retarded, in comparison with the apex beat. It is not rapid. Traube explained this fact in the following manner: On account of the aortic stenosis, the coronary arteries receive a diminished supply of blood, so that the heart is stimulated to less frequent contractions. The pulse is small, but incompressible and hard, on account of the left ventricular hypertrophy. It has an exquisitely tardy quality, *i. e.*, it is elevated very slowly and also sinks slowly into the condition of systole (Fig. 13). When the patients have a resistant, heavy-

FIG. 13.



Pulse curve in stenosis of the aortic orifice. After Marey.

ing apex beat, the difference between the quality of the apex beat and the smallness of the pulse should rouse a suspicion of aortic stenosis.

**PERCUSSION.**—This usually reveals slight enlargement of the area of cardiac dulness in a direction from above downwards and to the left.

**AUSCULTATION.**—A systolic murmur is heard over the heart with the greatest intensity in the second right intercostal space. It is very often characterized by a singing, whistling, or musical quality, is conveyed to the other valvular orifices, to the carotid, even to the head, and is also heard upon the posterior surface of the thorax, and occasionally at some distance from the patient. The degree of stenosis cannot be inferred from the intensity of the murmur.

The second (diastolic) aortic sound is very feeble, or entirely absent; if aortic insufficiency is also present, a diastolic murmur may be looked for. The second sound also grows feebler over the apex.

Unless special complications are present, the other heart sounds are unchanged, except that they are sometimes masked by the loud systolic murmur.

On auscultation of the carotid, we find, almost without exception, a systolic murmur which is conveyed from the aortic orifice. A second sound is almost always absent.

### *c. Mitral Insufficiency.*

In mitral insufficiency it becomes possible, during systole of the left ventricle, for blood to regurgitate from the latter into the left auricle. The left auricle, therefore, receives the regurgitated blood in addition to the normal amount from the right ventricle, and consequently becomes dilated.

As a result of the regurgitation of blood, symptoms of stasis become noticeable in the domain of the pulmonary veins. The stasis is propagated to the pulmonary capillaries and artery, and even to the right ventricle, so that all these vascular districts undergo dilatation. As the stasis implies elevation of blood pressure, hypertrophy of the right ventricle must follow the dilatation, *i. e.*, the right ventricle assumes



compensation for the valvular lesion. If the compensation did not develop, the stasis would be propagated into the right ventricle and the venæ cavæ, and the entire circulation would be disturbed, inasmuch as the venæ cavæ would be distended with blood, while the outflow into the aorta would be abnormally small on account of the partial regurgitation, and the tension in the arterial system would be unusually low.

Dilatation and slight hypertrophy of the left ventricle are associated not infrequently with the changes just described. The former would be expected so much more readily the more marked the mitral insufficiency, and the greater the dilatation and hypertrophy of the right ventricle. The cause may be sought for in the fact that the hypertrophy of the right ventricle gives rise to increased pressure in the domain of the pulmonary artery, and then in the pulmonary capillaries and veins. As the blood, therefore, flows under increased pressure from the pulmonary veins into the left auricle, then into the left ventricle, these parts must first undergo dilatation and then hypertrophy.

The murmur produced as the result of the valvular lesion arises in the left auricle; for, when the blood flows from the contracting left ventricle through the insufficient mitral valve into the relatively wide auricle, a whirl of blood and a murmur will be produced in the latter. These processes are favored by the fact that two currents of blood come in contact with one another, viz., that regurgitated into the left auricle and that entering from the pulmonary veins.

**INSPECTION.**—The cardiac region is prominent in many cases, even when the right ventricle alone is hypertrophied. The apex beat may be in the normal situation; when dilatation or hypertrophy of the right ventricle is very marked, the apex beat is situated outside of the left mammary line. If the left ventricle is hypertrophied and dilated, the apex is also lower than normal, and may even extend to the axillary line.

The diffuse cardiac impulse is sometimes noticeable even to the right of the sternum. This is chiefly the result of dilatation of the right heart, while hypertrophy of this part gives rise to vigorous elevation of the lower part of the sternum. This is conveyed not infrequently to the epigastrium which undergoes visible pulsating movements.

In some cases systolic pulsation is visible in the second left intercostal space. This occurs when the pulmonary artery is strongly dilated, and lies immediately adjacent to the chest wall after having pushed away the anterior median border of the left lung. In this situation, also, we observe occasionally during diastole a very short, quick pulsation, which corresponds, on palpation, to a short diastolic beat, and, on auscultation, to an intensified diastolic sound over the pulmonary artery. These phenomena result from the increased pressure in the pulmonary artery and the consequent hypertrophy of the right ventricle:

**PALPATION.**—In many cases a systolic purring thrill is felt over the apex. It is often not present unless the heart's action is excited (rapid walking, etc.). The thrill often becomes more distinct in left lateral decubitus.

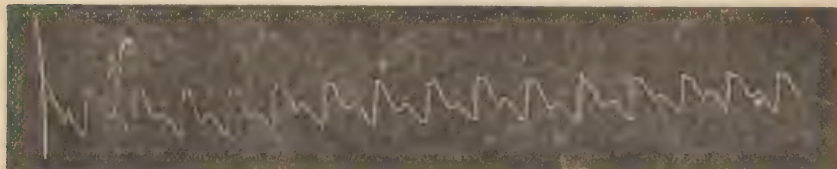
The diffuse apex beat is felt very far to the right, and an extremely active systolic impulse is often felt over the lower half of the sternum. In the second left intercostal space the palpating finger often feels a short, clicking impulse (particularly on deep pressure) alternating with the apex beat. This corresponds to the abnormally vigorous diastolic closure of the pulmonary semilunar valves. If the index finger of the right hand is placed over the apex, and that of the left hand in the

second left intercostal space, each will be struck alternately by the impulse.

The radial pulse is often normal, and is not always irregular, as Marey believed. Arrhythm of the pulse may be absent even when compensation has been disturbed.

The slight tension in the aortic system is shown by the marked elevation of recoil in the pulse curve, while the distinctness of the elevations of elasticity is impaired. As a matter of course, this is only true of uncomplicated and uncompensated cases (Fig. 14). But the pulse curve is not so characteristic as to enable us to recognize the valvular lesion.

FIG. 14.



Pulse curve of the right radial artery in mitral insufficiency. Girl, æt. 16 years.  $r$ , elevation of recoil.

**PERCUSSION.**—When the dilatation and hypertrophy affect only the right ventricle, the area of dulness increases chiefly in width, and assumes a roundish quadrilateral shape. The greater area of dulness extends beyond the right edge of the sternum, and the right border of cardiac resistance is more than four cm. from the right edge of the sternum at the level of the fourth costal cartilage.

If the left ventricle is also affected, the area of dulness increases in all directions, particularly from above downwards and to the left.

**AUSCULTATION.**—A systolic murmur is heard at the apex, and is conveyed very often to the tricuspid and pulmonary valves, more rarely into the aorta.

It is sometimes louder over the pulmonary orifice than the apex. A correct explanation of this fact was offered by Naunyn. He showed that the left auricular appendage surrounds, to a certain extent, the origin of the pulmonary artery, and thus may convey the murmur to the anterior wall of the thorax. It is sometimes conveyed a great distance, being heard over the thorax posteriorly or in the hepatic and splenic regions.

Sometimes a systolic murmur is heard only occasionally, particularly when the heart's action is excited, and it may disappear for a long time after intercurrent diseases and conditions of feebleness. This is owing to the fact that the development of whirling movements in the blood presupposes a certain rapidity of the current. The murmur may also be heard only in certain positions of the body, or be intensified in them. It follows, therefore, that the autopsy sometimes reveals the existence of mitral insufficiency, although no endocardial murmur was heard during life.

A systolic sound is almost always heard at the apex, in addition to the murmur, and the sound and murmur are usually produced at the same time. We may not conclude, therefore, that a leaflet of the mitral valve is still capable of vibration and of producing a sound, because the latter may result from contraction of the left ventricle, or be conveyed from

the tricuspid valve. The systolic sound is especially distinct when the ear is removed a little from the stethoscope, whereupon the murmur disappears.

Intensification of the second (diastolic) pulmonary sound also possesses diagnostic importance. This is evidence of increased pressure in the pulmonary artery, and hypertrophy of the right ventricle. The sound is usually strikingly clear and clicking, and has been compared to the short stroke of a hammer. Matterstock states that it is often extremely distinct in the left subclavian and axillary arteries.

The other heart sounds may be entirely unchanged. According to Matterstock, systolic murmurs are often conveyed into the carotid and subclavian arteries.

Cases of pure mitral insufficiency are very rarely found on autopsy; it is almost always associated with mitral stenosis. The clinical signs of the latter affection, however, may be absent. Cases are observed not infrequently in which the symptoms of mitral insufficiency predominated in the beginning, and gave way later to the signs of pure mitral stenosis. This is explained on the theory that the ring of insertion of the valve retracts and grows narrower, so that the valve again proves sufficient, while symptoms of mitral stenosis make their appearance.

Some authors maintain that relative mitral insufficiency is very frequent. But this view is exaggerated, to say the least, if we bear in mind that the diagnosis of mitral insufficiency cannot be based solely on a systolic murmur at the apex, and dilatation of the right ventricle (which may occur in emphysema, anæmia, and fever, despite intact mitral valves); but that permanent intensification of the second sound is also necessary.

Andrews states that he has seen several cases of recovery.

#### *d. Mitral Stenosis.*

In mitral stenosis, the blood flowing from the left auricle into the ventricle during diastole meets with an abnormal resistance. Hence the filling of the left ventricle and aorta is unusually slight, and requires longer time than under normal conditions, so that the tension in the aortic system is diminished.

At the same time, stasis develops in the left auricle, giving rise to dilatation of this part. The stasis is then communicated to the pulmonary veins, capillaries, and arteries, and finally to the right heart, where it first gives rise to dilatation and then to hypertrophy of the right ventricle.

The chief difference between mitral insufficiency and stenosis is the condition of the left ventricle. In the former, dilatation and hypertrophy of the left ventricle may develop, but in mitral stenosis this ventricle diminishes in size, on account of the small amount and low pressure of the blood which it contains.

In addition, these valvular lesions are distinguished by their acoustic phenomena. At the beginning of diastole, when the blood has passed the narrow mitral slit, and entered the empty diastolic, wide left ventricle, a whirling movement of the blood, *i. e.*, a murmur, develops in the latter. As the filling of the left ventricle requires a long time, the murmur is likewise of long duration.

INSPECTION.—The visible changes coincide, in great part, with those of mitral insufficiency. These include prominence of the cardiac region, from dilatation and hypertrophy of the right ventricle; diffuse cardiac



impulse, extending far to the right; sometimes systolic and diastolic pulsatory impulses in the second left intercostal space, corresponding to dilatation of the pulmonary artery and intensification of the diastolic closure of the pulmonary valve. The apex beat is sometimes moved a little to the left and externally, because the right ventricle may undergo such a remarkable degree of dilatation and hypertrophy that the entire heart assumes a more horizontal position.

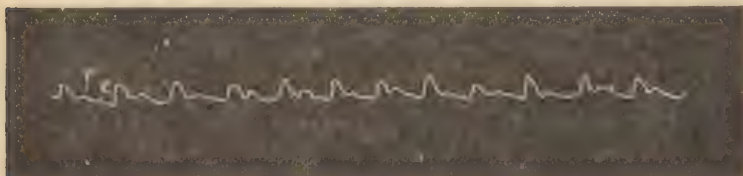
**PALPATION.**—The most important sign is a presystolic, rarely a purely diastolic, purring thrill; it is confined generally to the apex, is often more distinct in left lateral decubitus, but is not infrequently felt only at times, being increased or produced by increased action of the heart. The presystolic thrill ends with the elevation of the apex beat.

The thrill sometimes presents interruptions, being especially distinct at the beginning and end. Sometimes it is not felt until a very short time before the apex beat. It is easily distinguished from a diastolic thrill, because the latter precedes the apex beat by a distinct pause.

A short diastolic impulse is often felt in the second left intercostal space, as in mitral insufficiency.

The spread of the diffuse apex beat to the right, and its greater force, are readily felt with the hand.

• FIG. 15.



Pulse curve in mitral stenosis. After Riegel. *r*, elevation of recoil; *e*, elevation of elasticity.

It is evident in this curve that despite the slight elevation of the wave of blood, the elevation of recoil is well developed, while the elevations of elasticity become indistinct, or disappear. But the curve is not sufficiently characteristic to permit recognition of the valvular lesion from it alone.

**PERCUSSION.**—The area of cardiac dulness is enlarged towards the right, and assumes a more rounded, quadrilateral shape.

**AUSCULTATION.**—As a rule, a presystolic murmur is heard at the apex; this ceases at the beginning of the ventricular systolic sound. The systolic sound is followed by a pause, then comes the diastolic sound.

The murmur is often loudest at the beginning and end. As an endocardial murmur is so much louder the more rapid the current of blood, it is readily understood that in mitral stenosis the flow into the left ventricle is rapid at the beginning of diastole, because the ventricle is entirely empty; and that it is again accelerated at the end of diastole, because the contraction of the left auricle drives the mass of blood still remaining in it with greater rapidity into the left ventricle. The first half of the presystolic is sometimes entirely absent, and it is heard only during auricular systole, *i. e.*, at the end of ventricular diastole.

Much more rarely a purely diastolic murmur is heard over the apex. In these cases the last part of the presystolic murmur is absent, so that the murmur is separated from the systolic sound by a distinct interval.

It is characteristic of mitral stenosis that the intensity of the murmur

varies to a remarkable degree. It is sometimes present only when circulation is very active, as in bodily or mental excitement. It may even be absent at such times. Hilton Fagge found, upon autopsy, forty cases of, in part, marked mitral stenosis which never gave rise to a murmur during life.

Intensification of the second (diastolic) sound over the pulmonary artery is also important, as it is evidence of right ventricular hypertrophy. According to Matterstock, the sound is often conveyed with exceptional clearness into the axillary and subclavian arteries.

Both sounds over the aorta are extremely feeble because the aorta receives but little blood on account of the mitral stenosis.

If there are no special complications, the sounds over the tricuspid valve are unchanged, and the presystolic murmur is not infrequently conveyed here from the mitral valves.

The systolic sound is not infrequently intensified at the apex. Traube explained this on the ground that, as the result of the mitral stenosis, the difference in tension of the valve during the diastole and systole of the left ventricle is unusually great; on account of the slight and slow filling of the left ventricle with blood the leaflets of the mitral valve have not attained, at the end of the diastole, the degree of tension which exists under normal conditions.

The diastolic sound may be entirely absent at the apex. But if the pulmonary sound is greatly intensified, a clear, strong diastolic sound is also heard occasionally at the apex. Finally the diastolic sound may be conveyed from the tricuspid valve to the apex.

Sometimes a reduplicated diastolic sound is heard over the aorta and pulmonary artery; the first part corresponds to the closure of the aortic valves, the second and louder part to the closure of the pulmonary artery. According to Geigel, the pressure in the pulmonary artery differs from that in the aorta on account of the valvular lesion, and hence the valves of these vessels do not close at the same time. Closure occurs earlier in the vessel with lesser pressure (aorta). This phenomenon is generally absent in mitral insufficiency because the difference in pressure is not so marked in that lesion, particularly after dilatation and hypertrophy of the left ventricle have occurred. Systolic murmurs are often heard over the carotid and subclavian arteries.

Pure mitral stenosis is extremely rare, as it is almost always associated with mitral insufficiency. The symptoms of insufficiency very often predominate so that the stenosis is entirely concealed. Sometimes the insufficiency disappears and gives place to stenosis. This results from the retraction of the ring of the mitral valve, so that the originally insufficient leaflets again become sufficient. The prognosis is more unfavorable than in mitral insufficiency because the right ventricle alone is concerned in compensation.

### *e. Pulmonary Insufficiency.*

Pulmonary insufficiency gives rise to the same changes in the right ventricle that aortic insufficiency does in the left. As a part of the blood regurgitates into the right ventricle during its diastole, this portion must dilate in order to be able to contain the auricular blood plus the regurgitated blood. During the regurgitation a whirl of blood is produced in the right ventricle, corresponding acoustically to a diastolic murmur. In order that the right ventricle, during the next systole, should throw

the normal amount of blood in addition to the regurgitated blood into the pulmonary artery, it must perform more work and become hypertrophic. At each systole the pulmonary artery receives more blood than normal and becomes dilated.

**INSPECTION.**—The diffuse cardiac impulse extends to the right, and is visible outside of the right edge of the sternum. If the changes in the right heart are very considerable, the heart assumes a more horizontal position, and the apex beat may be situated a little outside of the left mammary line.

In some cases systolic pulsation has been observed in the second left intercostal space, corresponding to dilatation of the trunk of the pulmonary artery.

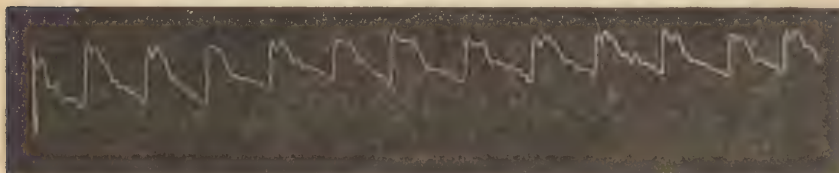
**PALPATION.**—Extension of the diffuse cardiac impulse is sometimes perceptible to the touch, though not visible to the eye. A diastolic purring thrill is often felt in the second intercostal space and over the lower part of the sternum.

In one case v. Bamberger felt systolic fremitus when the right ventricle, which could be reached in the epigastrium, was compressed with the fingers.

In the absence of complications, the apex beat and radial pulse are unchanged.

In a woman, *æt.* 31 years, I observed marked *pulsus tardus* (Fig. 16). The patient had acquired the valvular lesion in her ninth year.

FIG. 16.



Pulse curve of the right radial artery in insufficiency of the pulmonary valves in a woman *æt.* 31 years.

**PERCUSSION.**—Cardiac dulness and resistance are increased towards the right side.

**AUSCULTATION.**—A diastolic murmur is heard in the second left intercostal space. It is usually heard clearly over the lower half of the sternum, and is also conveyed to the aortic orifice, while it is very feeble or absent at the apex. The diastolic sound may be entirely absent over the pulmonary artery or is conveyed feebly to it from the aorta, or develops at the pulmonary orifice itself if some of the leaflets are still capable of vibration. As a matter of course, there is then a change in the second sound over the tricuspid valve, which is propagated from the pulmonary artery to the right ventricle; it is absent, or is replaced by a murmur, or is feeble.

The systolic sound over the pulmonary artery is not infrequently converted into a systolic murmur because the wall of the vessel undergoes irregular vibrations on account of excessive tension, but this does not justify us in diagnosing a complicating stenosis of the pulmonary orifice. The other heart-sounds may be unchanged.

v. Dusch showed that the diastolic murmur may be conveyed



from the pulmonary artery into the vessels of the neck, and this is probably more marked on the left side.

Pulmonary insufficiency is extremely rare; pulmonary stenosis also is usually present. As a rule, the lesion is congenital, more rarely it results from injury, arterio-sclerosis (Coupland), articular rheumatism in advanced age. Coupland's case occurred in a man æt. 75 years.

### *f. Pulmonary Stenosis.*

In pulmonary stenosis the blood meets with an abnormal resistance in passing, during systole, from the right ventricle into the pulmonary artery. In order to overcome this, the right ventricle must dilate, and then hypertrophy. At the same time the filling of the pulmonary artery proceeds very slowly, and under subnormal pressure. After the blood has passed through the narrow slit into the wide origin of the pulmonary artery, it undergoes a whirling motion, and gives rise to a systolic murmur.

INSPECTION.—There is generally a protrusion of the præcordium, as the disease is usually congenital. The protrusion is generally very distinct towards the left edge of the sternum (hypertrophy of the right ventricle). As a result of the hypertrophy, we sometimes notice diffuse impulse of the lower half of the sternum. The apex beat is generally feeble or absent, because that portion of the systolic recoil of the heart, which is normally the result of the systolic filling of the pulmonary artery, is abnormally slight.

PALPATION.—Systolic thrill is almost always noticeable. It is generally most distinct in the second left intercostal space, but is conveyed not infrequently over the entire cardiac region, and may even be felt distinctly upon the posterior surface of the thorax. Sometimes it is only present when the heart is unduly excited. Palpation of the diffuse cardiac impulse and apex beat gives results corresponding to those furnished by inspection. There are no characteristic changes of the radial pulse.

PERCUSSION.—Extension of cardiac dulness towards the right.

AUSCULTATION.—The characteristic auscultatory sign is a systolic murmur in the second left intercostal space. It is often very loud, and occasionally has a whistling and musical character. It is generally conveyed to the other cardiac orifices, and is heard not infrequently at a great distance from the cardiac region. The diastolic sound over the pulmonary artery may be entirely absent or it is extremely feeble. This results from the diminished pressure under which the blood, during systole, passes into the pulmonary artery. The other heart sounds may be unchanged, except that the second sound, as a matter of course, is feeble over the right ventricle, because it is conveyed from the pulmonary artery.

The systolic murmur is conveyed not infrequently into the subclavian, axillary, and carotid arteries. This occurs to a more marked degree in the arteries of the left side, into which the murmur may be exclusively conveyed. v. Dusch believes that the conveyance of the murmur into both carotids is evidence of the congenital character of the pulmonary stenosis. He believes that the conveyed murmur originates at the patent portion of the septum ventriculorum, and thence is readily conveyed into the aorta and higher parts.

Exceedingly loud, systolic, accidental murmurs are not infrequent

over the pulmonary orifice. If cyanosis and slight extension of the area of dulness to the right are also present, the diagnosis of pulmonary stenosis may be doubtful. As a rule, the accidental murmurs do not lead to the development of thrill, though this rule also presents exceptions. On the other hand, not every murmur due to pulmonary stenosis gives rise to thrill.

Acquired pulmonary stenosis is exceedingly rare; the lesion is usually congenital. The differentiation is not easy in all cases. Patients suffering from congenital stenosis are generally very cyanotic from childhood, but this symptom is not constant. In this form the autopsy generally reveals patent fetal channels of circulation (foramen ovale, septum membranaceum, septum ventriculorum, ductus arteriosus). But sometimes the foramen ovale remains open even in healthy individuals, and, on the other hand, Meyer has attempted to show that acquired pulmonary stenosis may cause reopening of the closed septum ventriculorum.

### *g. Tricuspid Insufficiency.*

In tricuspid insufficiency only a part of the blood flows into the pulmonary artery during systole of the right ventricle, while another part regurgitates into the right auricle. The right auricle, therefore, receives more blood than under normal conditions, and must dilate in proportion to the amount of blood regurgitated. It is evident, then, that the blood in the venæ cavæ will be under higher pressure as the result of stasis. The dilatation of the right auricle is followed directly by hypertrophy of its walls, and since, in consequence of this, the blood flows from the auricle under increased pressure during diastole of the right ventricle, the latter must also undergo dilatation and hypertrophy. As the blood regurgitates into the right auricle during ventricular systole, it there undergoes a whirling motion, which is increased by impact with the blood flowing from the venæ cavæ into the auricle. A systolic murmur develops accordingly in the right auricle. If the right ventricle and auricle possess sufficient power, a return wave of blood may be forced into the venæ cavæ at each systole, giving rise to venous pulse and hepatic vein pulse.

INSPECTION.—Præcordial prominence is observed not infrequently, particularly in congenital cases. The diffuse cardiac impulse usually extends to the right. Both phenomena depend on dilatation and hypertrophy of the right ventricle.

The venous pulse is a very important sign. It is most frequent in the cervical veins, appearing earliest in the internal jugular. It is sometimes confined to the lowermost portion of this vein, situated directly above the clavicle, only the bulb of the vein taking part in the pulsation. The pulsation is especially distinct when the bulb is situated unusually high. In other cases it is visible in the external jugular, thyroid, facial, temporal, frontal, auricular veins, the cutaneous veins of the upper and lower limbs, the thorax, and abdomen.

Venous pulse is a systolic distention and pulsation of the vein, passing from the heart to the periphery. In the cervical veins a double pulsation is observed not infrequently at each systole, the first elevation being smaller than the second. Venous pulse must be distinguished from other visible movements of the venous system. It is distinguished from respiratory distention of the veins by remaining unchanged during cessation of respiration. In some cases, movements are conveyed to the

cervical veins from the underlying arteries. The arteries should be compressed as far centrally as possible, whereupon the conveyed movement of the veins necessarily ceases. In addition, the cervical veins should be compressed about the middle of their course. If the movements were only conveyed, the vein collapses below the site of compression, and the pulsating movement disappears there, while it increases in the peripheral portion. In true venous pulse the wave passes unchanged with each systole from the heart to the site of compression.

The cervical venous pulse becomes more distinct in the horizontal position or when the head is lower than the trunk, and diminishes during deep inspiration. Geigel found that it is sometimes increased by compressing the inferior vena cava through the abdominal walls. It is sometimes present on the right side alone, on account of the more direct course of the right innominate vein and its branches.

Venous pulse arises from a return wave of blood, which is propelled feebly by the auricle, more vigorously by the right ventricle, into the venæ cavae and their branches. As long as the valves of the bulb of the jugular vein close, the wave meets with resistance at this point, and does not pass into the peripheral veins until these valves have become insufficient. Accordingly, cervical venous pulse is rather a sign of insufficiency of the valves of the bulb of the internal jugular than of the tricuspid valves, and it therefore occurs in other stases of the venæ cavae; for example, in those resulting from chronic pulmonary diseases. But it is particularly well marked in tricuspid insufficiency, because regurgitation of blood from the right ventricle occurs in that affection. The venous pulse disappears temporarily when the heart's action is weakened and the return wave no longer passes into the peripheral veins. It sometimes reappears after the administration of digitalis.

Similar conditions are found in the veins of the lower limbs. The return wave of blood in the inferior vena cava meets with resistance at the valves of the crural veins, near Poupart's ligament, and in the beginning we find a systolic impulse and pulsatory dilatation immediately below the ligament. After the valves have become insufficient, the pulsation extends into the saphenous vein.

The hepatic pulse depends on the same processes. As the wave of blood from the right heart passes through the inferior vena cava into the hepatic veins, a systolic, pulsating enlargement of the liver occurs, somewhat after the manner of an erectile tumor. It is sometimes observed before the jugular pulse, and, according to past experience, is met with only in tricuspid insufficiency. In a case of aortic insufficiency, O. Rosenbach observed hepatic pulsation as the result of abnormal filling of the hepatic artery.

Geigel showed that the inferior vena cava may become visible and palpable as a pulsating vessel to the right of the linea alba.

**PALPATION.**—The diffuse cardiac impulse usually may be followed far to the right; in a few cases, systolic thrill is felt over the lower part of the sternum.

Upon palpation of the veins of the neck, a short beat is felt occasionally over the bulb of the internal jugular vein, corresponding to the sudden unfolding of the valves. If the valves are insufficient, systolic thrill may be produced within the bulb. We can feel the pulsating veins fill at each systole and dilate laterally; indeed the feel of a pulsating artery may be simulated. The marked lateral distention during cardiac systole may be important in diagnosing conveyed from autochthonous venous



pulsation. All the phenomena are very often reduplicated, the feebler presystolic portion corresponding to auricular contraction, the more vigorous systolic portion to ventricular systole.

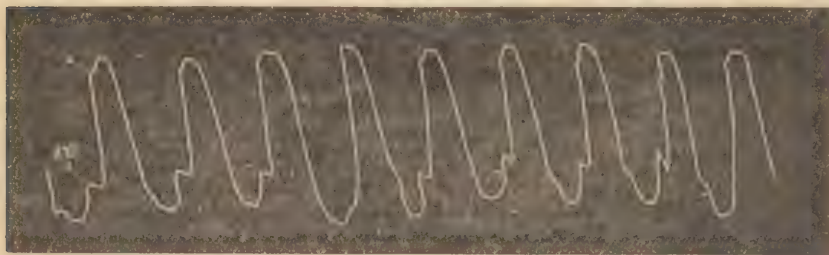
Palpable closure of the valves, systolic thrill, and marked pulsation may also be felt in the crural veins.

If the hands are applied anteriorly and posteriorly, or to the right and left upon the pulsating liver, it will be found that they are separated from one another at each cardiac systole. This distinguishes the hepatic pulse from the conveyed movement which is observed not infrequently in hepatic enlargement and vigorous pulsation of the abdominal aorta. If the fingers of one hand are laid in a circle upon the pulsating liver, it will be readily seen that they are not elevated, as in conveyed pulsation, but are separated from one another at each pulsation.

The venous and hepatic pulse are a little later than the apex beat of the heart, because the wave of blood requires a certain amount of time to reach the periphery.

The sphygmograph almost always shows anadictotism of the venous pulse in the neck, the small wave of the ascending line corresponding to the contraction of the right auricle (Fig. 17). Dicrotism is not infrequently visible in the descending line of the venous pulse curve; catadictotism rarely exists alone. The causes of the catadictotism are not known positively. Friedreich assumes that it results from a wave of blood which is reflected from the inner wall of the right ventricle.

FIG. 17.



Anacrotic venous pulse of the internal jugular vein. After Friedreich. *vc*, contraction of the auricle.

The hepatic pulse presents similar curves, and the pulse may be monacrotic, anadictotic, catadictotic, or anadictotic and catadictotic at the same time (Fig. 18).

FIG. 18.



Monacrotic hepatic venous pulse. After Friedreich.

The radial pulse is characterized by slight fulness and tension (Fig. 19).

**PERCUSSION.**—The cardiac dulness and resistance are increased towards the right, on account of the dilatation of the right auricle and ventricle.

**AUSCULTATION.**—A systolic murmur is heard over the tricuspid valves, and is usually loudest at the right edge of the sternum between the second and fourth right costal cartilages. It may be conveyed to all the cardiac orifices. The second pulmonary sound, and with it the diastolic sound over the right ventricle, are usually very feeble, because the pulmonary artery receives but little blood.

A short, clear, systolic sound (valve sound), is heard over the bulbs

FIG. 19.



Pulse curve in insufficiency of the tricuspid valves. After Marey.

of the jugular and crural veins, so long as the valves are sufficient. This sound may also be reduplicated and consist of a presystolic and systolic sound. When these valves become insufficient, a systolic or presystolic and systolic murmur develops, and if the tension of the walls of the vein becomes very considerable, a "venous wall sound" develops. Friedreich's observations show that insufficiency of the valves of the bulb may occur very rapidly (in a single night).

Isolated tricuspid lesions are rare. Among two hundred and thirty cases of valvular lesion, v. Bamberger found the tricuspid affected only in two. The lesion is generally congenital. Relative insufficiency of the tricuspid valves is frequent, particularly in the course of mitral lesions and chronic pulmonary diseases, and not very infrequent in aortic insufficiency. This complication may change the physical signs of tricuspid insufficiency. If the dilatation of the tricuspid orifice is very considerable, murmurs may be absent. Dieulafoy described a case of this kind in which the boundary between the right auricle and ventricle was almost entirely obliterated. This abolishes the conditions necessary to the development of whirling movements of the blood (sudden narrowing or widening of the current).

### *h. Tricuspid Stenosis.*

Pure tricuspid stenosis is hardly ever observed. It is almost always associated with insufficiency, and the valves of the left heart, particularly the mitral, are also usually affected. The signs of pure tricuspid stenosis must be described, therefore, on a rather theoretical basis.

In tricuspid stenosis the blood, in flowing during diastole from the right auricle into the ventricle, must overcome the resistance of the narrow slit. The consequent stasis in the right auricle gives rise to dilatation and subsequent hypertrophy of this part of the organ. While the pressure in the *venæ cavae* rises, it diminishes in the right ventricle and pulmonary artery, and at a later period the left ventricle and aorta must necessarily suffer. Cases have been reported in which, with the exception of the markedly dilated and hypertrophied right auricle, all other parts of the heart and the two main arteries were strikingly narrow.

After the blood of the right auricle, during diastole of the right ventricle, has passed the narrow slit of the tricuspid orifice, it undergoes whirling movements in the right ventricle, giving rise to a diastolic or presystolic murmur. The murmur remains absent if the current of blood is too slow to cause whirling movements, and such cases cannot be recognized.

The physical signs are: extension of cardiac dulness on the right side on account of dilatation of the right auricle; diastolic or presystolic murmur over the tricuspid valves; feeble second pulmonary sound on account of the slight filling of the pulmonary artery. Under certain circumstances, cervical venous pulse would be possible and would be chiefly presystolic.

### *i. Stenosis of the Arterial Coni.*

Stenoses are sometimes situated at the conus of the pulmonary artery or aorta. They usually result from fibrous myocarditic cicatrices, which, to a certain extent, have encompassed the conus like a ring. Such conditions are often congenital, but are sometimes acquired, for example, from injury in the cardiac region. Dittrich applied the term true cardiac stenosis to these changes. They are especially rare at the conus of the aorta. In general, they give rise to symptoms of valvular stenoses, but while the latter are almost always associated with insufficiency, the second sounds are clear in the former—a proof of the closure of the valves. According to Balfour, the diastolic sound is even intensified in stenosis of the conus pulmonalis. In addition, there are often evidences of a cardiac lesion which has existed, usually, since birth.

### *k. Combined Valvular Lesions.*

In combined valvular lesions, two things must be distinguished, viz., the combination of insufficiency and stenosis at the same valve, and the combination of lesions of two or more valves.

Combined insufficiency and stenosis of one valve is extremely frequent. Anatomically this constitutes the rule perhaps, but the clinical conditions are different, inasmuch as one or the other lesion predominates to such an extent that we are justified in speaking of insufficiency or of stenosis. This is especially frequent with regard to the mitral valve. Marked stenosis of this valve may be unattended with symptoms, so that the symptoms during life resemble those of insufficiency, but the autopsy shows that stenosis is the more marked lesion.

The diagnosis of combined lesions, particularly of the arterial orifices, requires great caution. Thus, in aortic insufficiency, the presence of a systolic murmur at the origin of the aorta does not justify the diagnosis of aortic stenosis, as the murmur may be produced by irregular vibrations of the walls of the vessel. Under such circumstances, the pulse merits the greatest attention, as it loses its bounding character the more marked the stenosis. This is also shown on the sphygmographic tracing (Fig. 20).

As a general thing, clinically pure insufficiency or stenosis is more frequent at the arterial orifices than at the auriculo-ventricular orifices. A combination of both is either present from the beginning or develops under observation. Sometimes pure insufficiency is present at the start, and then becomes associated with stenosis which finally persists alone.

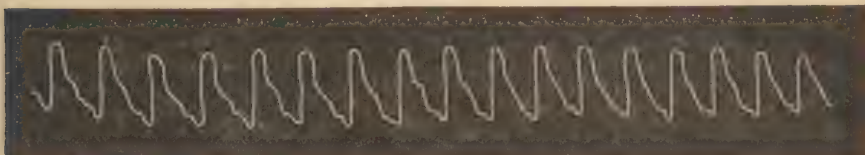
The combination of insufficiency and stenosis at the same valve is not an unfavorable event, for each lesion tends to eliminate the disturb-



ances produced by the other. This may be illustrated by the combination of aortic stenosis and insufficiency. During left ventricular diastole, the stenosis prevents the blood from regurgitating as freely through the insufficient valves as it would in the absence of the stenosis. In left ventricular systole, likewise, the effect of the insufficiency is diminished by the stenosis which is present. On the other hand, the regurgitation of blood which nevertheless does occur, does not permit a pure stenosis effect.

From an etiological standpoint, two forms of combination of valvular lesions must be distinguished, a mechanical and an anatomical combination. The mechanical combination refers almost exclusively to the relation between tricuspid insufficiency and mitral lesions. We have pre-

FIG. 20.



Pulse curve in insufficiency of the aortic valves with stenosis of the aortic orifice. After Rosenstein.

viously shown that mitral lesions are very often followed by relative tricuspid insufficiency. This is simply the result of gradual dilatation of the right ventricle and tricuspid orifice, so that the tricuspid valves no longer suffice to close the opening.

In the anatomical combination, endocarditic changes occur upon a number of valves. This is much more common anatomically than demonstrable clinically. Sometimes the different valves are affected from one and the same cause, sometimes the endocarditic process spreads gradually from one valve to the other. The spread of an aortic lesion to the aortic leaflet of the mitral valve is observed most frequently. In certain cases, endocarditic changes appear to be secondary to the excessive tension of individual valves resulting from previous lesions of other valves.

There is a large number of possible combinations, so that we must confine ourselves to furnishing a few illustrations.

The effects of a combination of valvular lesions upon the circulation are sometimes favorable, sometimes unfavorable. For example, aortic insufficiency and mitral stenosis constitute a favorable combination. For although the left ventricle receives less blood than normal on account of the mitral stenosis, this is counteracted as much as possible by the regurgitation of blood which occurs on account of the aortic insufficiency. At the same time, the slow and slight filling of the left ventricle prevents the excessive distention of the aortic system with blood during systole. On the other hand, the effects of the mitral stenosis on the right heart are diminished, for the reason that the dilated and hypertrophied left ventricle accelerates the flow of blood from the left auricle to a greater extent than would occur in the absence of dilatation of the left ventricle.

Among the unfavorable combinations is that of aortic stenosis and mitral insufficiency. The aorta receives an abnormally small amount of blood on account of the aortic stenosis. This amount is diminished still more because a part of the blood regurgitates into the left auricle on

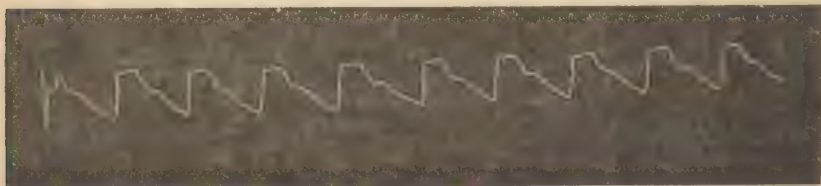
account of the mitral insufficiency, and, moreover, the regurgitation is especially great on account of the ventricular hypertrophy.

Very slight changes develop when all the cardiac orifices, except the pulmonary, are stenosed. The heart remains almost unchanged, and, in the main, slowness of the circulation seems to be the sole effect.

As a matter of course, a combination of valvular lesions can only be recognized by a combination of the corresponding physical signs. The diagnosis will be rendered much easier if the combination is of such a character that a systolic murmur develops at one orifice, a presystolic or diastolic murmur at the other. But if the murmurs occur at the same time, we are in danger of regarding them as autochthonous and vice versa, and in such cases we must pay special attention to the timbre, intensity, and propagation of the murmurs. Thus, autochthonous tricuspid and aortic murmurs persist as such at a certain distance to the right of the cardiac region proper, while sounds become audible if the murmurs referred to above have been merely conveyed. The reverse holds good concerning pulmonary and mitral murmurs. If we suspect a combination with mitral lesions, we must look for intensification of the second pulmonary sound, or, in stenosis, for a presystolic murmur. A combination with tricuspid lesions is recognizable by the venous pulse, but this is not a constant symptom.

The following pulse curve shows that in a combination of lesions of two valves—in our case aortic and mitral insufficiency—the characteristic form of the pulse is lost. It is readily seen that the curve no longer presents the characteristics of *pulsus altus*, such as occurs in pure aortic insufficiency, and is shown in Fig. 12.

FIG. 21.



Pulse curve of the right radial artery in insufficiency of the aortic and mitral valves. Woman æt. 36 years.

*The general symptoms of valvular lesions* are almost exclusively the results of changed circulation. So long as the valvular lesion is in the most perfect condition of compensation possible, these symptoms are absent or slightly marked; they become more prominent, and usually lead to a fatal termination, when the power of the heart muscle fails on account of fatty or fibroid degeneration, and thus allows the full effect of the valvular lesion to come into play. These conditions of disturbed compensation are the result of the excessive obstruction to the outflow of the blood of the venæ cavæ, which extends peripherally into the capillaries, and may even be propagated farther back into the aortic system.

Valvular lesions of the heart may give rise to disturbances in all the organs. Indeed, the symptoms on the part of one or the other organ are not infrequently so prominent that superficial examination, particularly of the heart, may lead to gross mistakes. Thus, the patient may

be supposed to suffer from pulmonary disease because he complains of dyspnœa, cough, and expectoration. In other cases, they suffer from violent gastric symptoms, and gastric ulcer may be suspected on account of severe gastralgia which results from the valvular lesion.

Certain general symptoms depend on the site of the valvular lesion. They occur chiefly in certain lesions, and have a sort of specific significance, though they do not depend exclusively upon such lesions.

1. *Subjective Symptoms*.—In well-compensated valvular lesions, subjective symptoms may be entirely absent, though this is generally true only of aortic insufficiency, in which the left ventricle assumes the compensation. This ventricle, on account of its large muscular mass, is capable of permanently doing an increased amount of work. Such patients may even undergo very considerable bodily exertion without disturbance.

In other cases, annoying palpitation, dyspnœa, and a feeling of oppression only appear after great bodily or mental excitement. This occurs after running, gymnastics, dancing, riding, occasionally after the use of tea, coffee, or tobacco, after a hearty meal, or in certain positions of the body. The left lateral decubitus is especially apt to give rise to dyspnœa and palpitation.

Finally, other patients are rarely free from subjective symptoms, and the individual is annoyed by the symptoms mentioned even when the valvular lesion is in a condition of relatively good compensation.

2. *Constitution*.—In valvular lesions acquired by adults, the constitution depends upon chance. If the lesion dates from childhood, the bodily development generally remains defective. Even in adults the nutrition is impaired the longer the valvular lesion exists, and particularly the oftener and the longer compensation has been disturbed. A very high grade of cachexia (cachexie cardiaque of Andral) may develop in consequence. We will here refer to a peculiar deformity of the fingers which is observed particularly when the valvular lesion has been acquired in early childhood. The ungual phalanges become swollen into a club shape, and the fingers may assume a claw-shaped position.

3. *Œdema*.—This is one of the most frequent and constant symptoms, and may be the result of various factors. The chief one is stasis *i. e.*, increased pressure in the venous system, which is necessarily continued into the lymphatic vessels, since the latter are taken up by the veins. Not infrequently there is a change in the constitution of the blood, which becomes poorer in solid constituents, particularly albumin, and thus changes the vascular walls, *i. e.*, makes them more porous.

Œdema generally develops first in the subcutaneous cellular tissue, and earliest in the lower limbs, as in other forms due to stasis. In the beginning the œdema appears around the ankles, and is often present only during the day, disappearing at night in the recumbent posture. The œdema becomes permanent at a later period, extends throughout the lower limbs, then extends to the genitals, abdominal and thoracic walls, finally to the integument of the upper limbs and face. If the patients are in the habit of lying chiefly on one side, the œdema accumulates in preat part, sometimes almost exclusively, on this side from purely mechanical causes.

Œdema enfeebles the economy on account of the loss of nutritive fluid. By compression of the cutaneous vessels it increases the obstruction in the arterial system. Finally, it may give rise to very dangerous, local changes in the skin. In some cases, erythema or fissures appear



upon the surface of the swollen skin, and give vent to the fluid. The skin then becomes inflamed very generally, and erysipelatous changes arise. In other cases, vesicles form, burst, and allow the escape of serous fluid. These are followed sometimes by ulcerative and gangrenous processes.

Next to the subcutaneous tissue, the œdema involves the serous cavities. Ascites generally develops first, and is followed by hydrothorax, hydropericardium, and œdema into the meningeal spaces and cerebral ventricles. Ascites, hydrothorax, and hydropericardium necessarily increase the resistance to the failing heart's action, while the danger of paralysis is not inconsiderable when changes occur within the cranium.

4. *Skin*.—The integument almost always has a cyanotic color. This is so much more marked the more the pulmonary circulation is affected from the start by the valvular lesion, and it is generally more marked in mitral than in aortic lesions. Especially high grades of cyanosis are often observed in pulmonary lesions. The cyanosis always increases when the disturbances of compensation become more marked. If the cyanosis is slight, the layman not infrequently regards the bright red color of the face and visible mucous membranes as a sign of remarkably good health, and it is only by the experienced eye that the ominous significance to the bluish red is recognized.

Meshes of dilated vessels appear not infrequently upon the cheeks and nose, and are followed occasionally by inflammatory and hyperplastic processes in the skin, which lead to the development of acne rosacea.

The large cutaneous veins are not infrequently very strongly dilated, and they may present varicose dilatations, which may be associated under certain circumstances with other morbid conditions, for example ulcerations.

In addition to cyanosis, the skin may present the signs of icterus. Sometimes it is confined to the conjunctiva, in other cases it extends over the entire integument and, associated with the cyanosis, imparts to the skin a dirty yellow or distinctly green color (*icterus viridis*). The jaundice generally develops gradually and lasts for a long time. In rarer cases it develops with great suddenness and intensity and is the result of embolic processes in the hepatic artery.

Cutaneous hemorrhages are sometimes observed. These may be embolic in their origin (although capillary emboli are not frequent in chronic endocarditis) or they may be true cutaneous hemorrhages. In the former event, we must pay attention to the bright centre, which corresponds to the site of the embolus. If the patients are very feeble, a sort of hemorrhagic diathesis sometimes develops. Small extravasations appear upon the skin and mucous membranes for weeks and months, so that the symptomatology is similar to that of *morbus maculosus Werlhofii*. Some authors have erroneously spoken of a scorbutic dyscrasia in such cases.

If œdema of the skin relapses frequently, thickening of the subcutaneous connective tissue may ensue, and the symptoms of elephantiasis appear.

5. *Bodily Temperature*.—The temperature of the body is normal in many cases, subnormal in others. The limbs often feel ice cold if stasis is manifested in them by cyanosis and œdema. This is evidently the result of slowness of the circulation of blood.

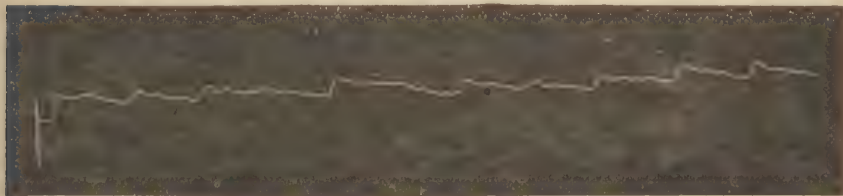
Intercurrent rise of temperature is often observed in embolic processes

in internal organs. Elevation of temperature also occurs, though not constantly, in hemorrhagic infarctions into the lungs.

6. *Pulse*.—The pulse presents certain general characteristics, in addition to those which are peculiar to some valvular lesions. When disturbances of compensation occur, the pulse usually becomes very frequent and irregular in rapidity and strength. It is often intermittent, *i. e.*, not every contraction of the heart is sufficient to drive the blood into the radial artery, so that there are a greater number of heart beats than pulse beats. At the same time the pulse loses in strength and usually feels soft and less full.

The irregularity of the pulse is also shown by the sphygmograph in conditions of disturbed compensation.

FIG. 22.



Irregular and intermittent pulse in uncompensated mitral insufficiency in a man *æt.* 27 years.

7. *Heart*.—One of the most frequent symptoms is palpitation of the heart, which may be either subjective or objective. The acceleration of the cardiac movements is demonstrable objectively only in the latter form, while in the former the annoying sensation of palpitation is felt, though it does not appear to arise from excited action of the heart. Sometimes the palpitation is almost uninterrupted, sometimes it is produced by bodily or mental exertion, sometimes it is the sign of threatening or fully developed disturbance of compensation. It is accompanied occasionally by a feeling of peculiar tension and constriction in the region of the heart, sometimes there are extremely distressing and painful sensations which radiate from the præcordial region into the left arm, the neck, and umbilicus, and correspond almost exactly to the stenocardic attacks which will be described later. These symptoms appear with relative frequency in aortic insufficiency, and seem to be the result in some cases of mechanical irritation of the cardiac plexus by the dilated aorta. Michael Peter claims to have observed changes in the ganglion cells of this plexus.

If the circulation is very slow and changes have occurred in the endothelium of the endocardium, thrombotic deposits may form within the cardiac cavities, generally in the auricular appendages or among the trabeculæ of the ventricles. These possess grave significance. By their excessive development they may narrow adjacent orifices; in other cases particles break off, pass from the left heart into the peripheral arteries and give rise to embolic occlusion, or from the right heart into the lungs and cause sudden death if the main branches of the pulmonary artery are obstructed.

In rare cases death results from rupture of the heart, in others it seems to depend on sudden paralysis of the heart muscle.

Disturbances of compensation are manifested sometimes by peculiar changes

in the cardiac movements. The latter are not entirely irregular, but there is an unusual rhythm of the heart's action (allorhythm). It is observed with relative frequency in mitral lesions, particularly insufficiency, especially when they are associated with relative insufficiency of the tricuspid valves. It is found that two complete cardiac movements follow rapidly upon one another and are separated by a long pause from the next pair, *i. e.*, there is bigeminy of the heart's action. The apex beat, heart sounds, and murmurs of the second contraction are shorter than those of the first, and in addition the radial pulse is absent or only indicated. In the former event, accordingly, auscultation shows twice as many cardiac contractions as can be counted at the radial pulse. In the patient whose curve is shown in Figure 23, the phenomenon lasted a week, alternated with regular and irregular movements of the heart, and then disappeared altogether, at least during a period of observation lasting four months.

FIG. 23.



Cardiac bigeminy in a man, *æt.* 33 years, suffering from insufficiency of the mitral valves.

Probably many cases of Leyden's hemisystole should be included in this category. According to Leyden, hemisystole is that form of cardiac contraction in which both ventricles, and then the right ventricle alone, contract alternately, so that, in tricuspid insufficiency, intermittent radial pulse and venous pulse of the neck, and venous pulse of the neck alone are observed. It is evident that this must also occur in bigeminy if the second (total) contraction of the left ventricle possesses too little force to produce a radial pulse, while the right ventricle gives rise to the easily produced venous pulse of the neck.

8. *Blood-vessels.*—Among the changes in the vessels, embolic and thrombotic processes attract the chief attention. If the emboli are derived from the right heart, they pass into the branches of the pulmonary artery, and give rise to hemorrhagic infarctions. If the left heart gives rise to emboli by rupture of pieces of the valves, or separation from thrombi, the arteries of the limbs, spleen, kidneys, brain, are most frequently affected, more rarely the mesenteric, hepatic, retinal, or small cutaneous arteries.

Emboli in the arteries of the limbs are manifested by sudden pains in



the limb, a feeling of weakness, coldness, stiffness, diminution of sensibility, and paræsthesia. The limb is pulseless below the site of embolism. The symptoms become less marked, and gradually disappear, when the collateral circulation restores the connection of the vessel above and below the site of embolism. If the development of collateral paths remains absent or is insufficient, the limb becomes gangrenous. If the lower limb is affected, as is generally the case, dry gangrene generally develops on the toes and dorsum of the foot, while vesicles and symptoms of moist gangrene appear upon the legs. The embolus is usually unilateral, the left lower limb being affected most frequently. This is owing to the fact that the left iliac is given off from the aorta in a straighter line than the right iliac, and the embolus carried along in the current of blood follows, in the main, a straight line. If both lower limbs are affected, the site of the embolus must generally be looked for in the lower part of the aorta and above its final bifurcation. But there are also cases in which each iliac is occluded by an embolus.

In embolism of the arteries of the upper limbs, the difference in the strength of the pulse on the two sides is often maintained during life. By careful search for the place at which the pulse becomes equal on the two sides, we can determine the position of the embolus. It must be remembered, however, that the radial artery occasionally runs an anomalous course, and may thus give rise to diagnostic errors.

Embolism of the splenic artery is shown by sudden chill with subsequent rise of temperature and sweating; vomiting occurs occasionally. In addition, there are pains in the splenic region and demonstrable acute enlargement of the spleen.

Chill, elevation of temperature, sweating, vomiting, are also observed in renal embolism. These symptoms are associated with pains in the region of the kidneys and bloody urine. Small emboli may be entirely latent.

Cerebral emboli are situated generally in the domain of the left carotid, because this passes upwards somewhat in the direction of the arch of the aorta, while the right carotid is given off at a right angle from the aortic arch.

Frequently, though not constantly, the emboli lodge in the left middle cerebral artery, giving rise to an easily recognized clinical history, which is characterized in the main by right hemiplegia and aphasia. Cerebral embolism is attended generally by an apoplectic seizure; the patients suddenly fall unconscious, and, upon the restoration of consciousness, are found to be paralyzed on one side.

Emboli in the retinal arteries are readily recognized with the aid of the ophthalmoscope. They are not frequent, first, because the ophthalmic artery is given off from the internal carotid at a right angle, and also because the central artery of the retina is given off at a right angle from the ophthalmic artery. This lesion is recognized functionally by the sudden blindness. The ophthalmoscope shows striking narrowness of the arteries which, at times, may disappear almost entirely. The macula lutea usually appears as a cherry-red spot. The retinal veins are often narrowed, and, in some cases, it is hardly possible to distinguish them from the arteries. The column of blood in them sometimes appears interrupted, and A. v. Graefe observed in it arrhythmical and intermittent progression towards the optic papilla. Near the latter the retina is whitish and cloudy, and atrophy of the optic nerve may follow.

Virchow and Knapp have described occlusion of individual branches

of the retinal arteries. Emboli of the ciliary arteries have also been observed.

Embolism of the superior or inferior mesenteric artery is manifested by sudden abdominal pain, peritonitic symptoms, bloody passages, and usually rapid and fatal collapse. Embolism of the hepatic artery may give rise to the symptomatology of acute yellow atrophy of the liver.

Thrombi sometimes form in the large venous trunks. If particles break off from them, they are carried into the right heart, and thence into the lungs. Death ensues if a large branch of the pulmonary artery is occluded. M. Seidel reports a case of aortic stenosis and insufficiency, in which embolism of the trunk of the pulmonary artery and sudden death followed thrombosis of the common iliac vein.

We will refer later to the excessive brittleness of the small vessels in consequence of fatty degeneration or arterio-sclerotic changes.

9. *Respiratory Organs.*—The majority of patients complain of a feeling of want of breath. This is sometimes constant, sometimes appears after bodily or mental excitement in conjunction with palpitation, and sometimes it appears only at certain periods, particularly during the night.

The causes of the dyspnoea are not always similar, and in not a few cases a number of causes co-operate. In the first place, there is a mechanical narrowing of the lungs by the enlarged heart, and this affects chiefly the lower lobe of the left lung. This mechanical disturbance increases when the space occupied by the lungs is still further encroached upon by hydrothorax and ascites. Heavy meals also exert a great influence on the occurrence of dyspnoea.

In many cases the cause must be sought in the slowness of the lesser (pulmonary) circulation, which is partly dependent on the heart itself, and is partly favored by the fact that the structurally changed lungs no longer fulfil their purpose of stimulating the circulation by inspiration and expiration. Bronchial catarrh and extensive hemorrhagic infarctions in the lungs may also favor the development of dyspnoea. Finally, King and Friedreich have described cases in which compression of the left main bronchus, by the enlarged left auricle, had impaired the activity of the left lung to a considerable extent. This condition would be shown by the fact that the part played by the left lung in inspiration is diminished and delayed, by the feeble respiratory murmur and the murmur of stenosis, and if the compression of the bronchus is very considerable, we should even expect inspiratory retraction of the intercostal spaces and diminished vocal fremitus.

The patients suffer very often from bronchial catarrh, particularly when the mitral valve is affected. It sometimes continues for months and years, exacerbations and remissions following one another in rapid succession.

The same causes to which the bronchial catarrh is owing, viz., the abnormal elevation of pressure in the pulmonary veins, also gives rise, in many cases, to hæmoptysis. More frequently, however, it seems to follow embolic processes in the branches of the pulmonary artery. The hæmoptysis may be very considerable, last for several days, and, though rarely, lead directly to a fatal termination. This form of hæmoptysis is termed hemorrhagic infarction. Many patients outlive a large number of infarctions, and, according to Bamberger, they are frequent in aortic stenosis and also in mitral stenosis.

If the extravasations into the pulmonary parenchyma are not large,

they may remain latent. A peculiar brownish-red sputum may appear after a certain lapse of time, and resembles to a certain extent the rusty sputum of pneumonia. This continues occasionally for weeks and months, and evidently owes its color to changes in the blood pigment of the extravasated blood-corpuscles. Microscopical examination of the sputum shows peculiar large, finely granular cells, in which we recognize, not infrequently, a large, elongated oval nucleus. These are sometimes of a diffuse yellow color, sometimes filled with brown or yellow pigment granules, in addition they contain fine brown needles and small rhombic plates. The cells in great part are epithelium cells from the pulmonary alveoli, although a part, according to Rindfleisch, are derived from amœboid round cells. In some places, free granules, needles, and tables of hæmatoidin are also found in the sputum. When preceded by hæmoptysis, the sputum may assume a brownish-red color after a number of days, and microscopical examination shows the appearances just described. Sometimes a number of days elapse during which the sputum is entirely colorless, and then becomes brownish-red.

French writers maintain that the hæmoptysis of hemorrhagic infarction may be distinguished from that of primary diseases of the lung by an examination of the sputum. Guéneau de Mussy says that in forty cases he detected a peculiar acid, garlic-like odor reminding one of tincture of radish, and maintains that he can recognize a latent hemorrhagic infarction by this odor. Although I have made careful investigations, I have never had a similar experience. Hyde Salter attaches great diagnostic importance to the fact that in hæmoptysis of heart disease the blood is clotted and bluish-black.

Pulmonary œdema is a frequent cause of death in heart disease. It is easily recognized by the widespread crepitant râles and the serous expectoration. If hemorrhagic infarctions have developed some time previously, the expectoration is brownish-red from admixture with blood pigment cells (from œdema of the lungs). The experiments of Cohnheim and Welch have rendered it probable that this is a pure stasis œdema which develops when the left ventricle is paralyzed while the right continues to act.

Inflammation of the lungs also carries off no small proportion of heart cases, and valvular lesions undoubtedly offer a marked predisposition to inflammatory changes in the respiratory apparatus. Œdema of the glottis is also a cause of death in individual cases.

Epistaxis must also be included among the affections of the respiratory tract. Sometimes it occurs unexpectedly, while in other cases it is preceded by symptoms of a rush of blood to the head (dizziness, ringing in the ears, spots before the eyes, a feeling of dulness, etc.). In the latter event the symptoms of congestion not infrequently subside after the epistaxis. If the hemorrhages recur rapidly or if they continue for a long time and are checked with difficulty, very grave conditions of weakness develop.

10. *Digestive Organs.*—Disturbances of appetite and digestion occur very frequently. They are the result of stasis catarrhs of the gastrointestinal mucous membrane, which may increase to erosions and hemorrhagic infiltrations of the mucous membrane. Heavy meals exercise a bad influence on the heart's action, and are followed not infrequently by palpitation and anxiety. The bowels are usually constipated; diarrhœa is less frequent. The evacuations are sometimes bloody, generally from extravasations upon the intestinal mucous membrane or from hemorrhoidal hem-



orrhages, rarely from embolism of the superior or inferior mesenteric artery. Vomited matters, which are generally mucoid, sometimes contain blood. Persons suffering from valvular disease of the heart are often affected by varicose dilatations of the hemorrhoidal veins, and these may give rise to very great annoyance. If signs of dissolution of the blood make their appearance, extravasations will also be found upon the mucous membrane of the mouth. We have previously referred to the cardialgia which may be erroneously attributed to a gastric affection.

Enlargement of the spleen is much rarer than that of the liver, and it almost seems as if the circulation of the portal vein is capable of diminishing the stasis in the inferior vena cava and keeping it remote from the spleen.

11. *Abdominal Glands.*—The liver is more or less enlarged in many cases. The patients also complain not infrequently of a peculiar feeling of tension, pressure, and weight in the right hypochondrium. The liver can usually be felt at its lower border; its edge is rounded, peculiarly firm, soft, and resisting. If the valvular lesion has existed for a long time, the liver may again diminish in size, but its consistence grows firmer and the lower border sharper.

12. *Urinary and Genital Apparatus.*—The amount and constitution of the urine enable us to judge of the severity of the disturbances of compensation. The more the pressure in the aorta is diminished and the higher it is in the venæ cavæ, the more the urine assumes the character of stasis urine. The quantity diminishes considerably, the color is strikingly red, the solid constituents and specific gravity increase. On account of its great concentration, the urine, on growing cold, not infrequently deposits uric acid salts (urates) in the form of a reddish sediment looking like brick-dust. The urine is proportionately rich in urea, but the amount passed in twenty-four hours is diminished; the excretion of uric acid is increased, even as much as eightfold, according to Daremberg. If œdema is present, the amount of chloride of sodium in the urine is small. It often contains a small amount of albumin, hyaline casts, and red blood-globules. Hæmaturia is not often observed, and is usually the result of inflammatory changes in the renal parenchyma or of embolic processes.

Sometimes the urine presents the characters of chronic Bright's disease; we have previously mentioned that valvular lesions may develop during the course of the latter disease. Catarrhal changes in the mucous membrane of the pelvis of the kidneys, ureters, and bladder may be shown by the copious admixture of mucus and pus corpuscles with the urine, but this is not very frequent.

Women suffer not infrequently from a mucous and muco-purulent discharge from the genitals. Menstruation is generally profuse, or pseudo-menstrual hemorrhages occur; dysmenorrhœic conditions are less frequent. Varicocele or hydrocele may develop in males.

13. *Eye.*—In addition to embolic processes, the retina may also present extravasations of blood with inflammatory appearances (retinitis hemorrhagica). Hemorrhages may also occur in the vitreous body. These extravasations are sometimes the result of excessive stasis, sometimes they result from hypertrophy of the left ventricle and changes in the walls of the vessels.

Icteric discoloration of the conjunctiva has been previously referred to; hemorrhages under the conjunctiva may also occur.

14. *Nervous System.*—Symptoms on the part of the central nervous

system are not rare; they may be the result of anæmic, hyperæmic, embolic, or hemorrhagic conditions.

Cerebral anæmia is manifested chiefly by syncope.

This is observed not infrequently in aortic stenosis, because this lesion prevents the normal filling of the aorta. The occurrence of syncope must be looked for particularly when the erect position is suddenly assumed after the patient has long been in the recumbent posture. This may also happen in aortic insufficiency when the left ventricle has undergone fatty degeneration.

Cerebral hyperæmia is manifested by dizziness, dulness in the head, spots before the eyes, tinnitus aurium, etc.

The symptoms of cerebral embolism have been referred to casually. Embolism of the left middle cerebral artery gives rise to right hemiplegia and aphasia. But it must be remembered that temporary aphasic symptoms may occur without embolism, and appear to be due rather to intercurrent circulatory disturbances.

Gryan reports, in a case of mitral insufficiency and stenosis, the sudden occurrence of aphasia which disappeared with equal suddenness at the end of a week. At the autopsy, which was held seven weeks later, no anatomical changes were found in the brain.

The frequency of cerebral hemorrhage as the result of valvular lesions has been greatly overestimated. There is indeed a predisposition to hemorrhage, on account of the fatty degeneration and arterio-sclerotic changes of the smaller vessels in the brain. Rupture of intact vessels, as the sole consequence of excessive blood pressure, probably never takes place. If the valvular lesion is congenital, or begins in early childhood, the mental development may suffer. The patients are apathetic, ill-natured, learn with difficulty, and live a retired life. Hypochondriacal conditions develop not infrequently in later life. According to d'Astros, patients suffering from mitral lesions have a tendency to melancholic conditions, those suffering from aortic lesions seem more predisposed to conditions of excitement.

Well-marked insanity is rarely observed in patients suffering from heart disease. Temporary delirium and maniacal attacks sometimes occur, have a tendency, according to Peter, to appear especially at night, and have a bad prognostic significance. They have been attributed to temporary conditions of cerebral congestion. Epileptiform attacks have also been described a number of times.

The central nervous system sometimes has a special significance with regard to the termination of the disease, inasmuch as death sometimes appears to result from paralysis of the nerve centres. As a rule, however, it is difficult to decide whether death is caused by paralysis of the heart or of the central nervous system.

15. *Relations to other Diseases.*—All complicating diseases present a specially grave significance in individuals who suffer from valvular lesions. This is particularly true of febrile diseases. It is known that the nutrition of the heart muscle suffers under the influence of elevated bodily temperature. If the febrile nutritive disturbance may go so far in the healthy heart as to render it incapable of function, how much more readily is this done in valvular lesions, in which the maintenance of life depends on the increased work of the heart.

All intercurrent diseases of the respiratory organs also assume a grave significance. They assign to the right heart a greater amount of work even than is rendered necessary by the valvular lesion, and it may very

easily happen that the heart is no longer able to meet the increased demand for work.

We will here refer to the relations between valvular lesions and pregnancy. Pregnancy and child-birth may be unexpected causes of death in women suffering from valvular lesions, and mitral lesions have an especially unfavorable influence. The connection is not the same in all cases. Loeblein showed that the processes during child-birth have a great tendency to cause acute relapses of endocarditis, thus constituting a fruitful source of danger. If fatty degeneration of the heart has occurred as the result of the valvular lesion, rupture of the heart and sudden death may occur during delivery. Spiegelberg attaches great importance to the fact that the interpolation of the placental circulation imposes upon the heart an increased amount of work, which it is sometimes unable to meet, while it is suddenly relieved immediately after delivery.

Mention must now be made of diseases, some of which occur very frequently in valvular lesions, while others are hardly ever observed.

Chorea is said to occur with special frequency in valvular lesions, and a great influence upon the development of this disease is attributed to mitral lesions. French and English authors have greatly overestimated the frequency of this connection, but there can be no doubt that such a relation does exist. Some explain the relation by embolic changes in the central nervous system, others by reflex influences (irritation of the phrenic nerve by the enlarged heart).

According to a few observations, erythema nodosum and peliosis rheumatica may be the result of embolism of the cutaneous arteries following endocarditis, but these cases are by no means conclusive, and at all events they constitute the exception rather than the rule.

Beyer and Rosenbach observed the frequent association of aortic insufficiency and tabes. This has been corroborated by Grasset (fifteen cases) and Letulle.

It was formerly held that valvular lesions exclude the development of typhoid fever, cancer, and phthisis. Recent statistics have disproved this proposition, so far as regards typhoid fever and cancer. In the next place, Frerichs proved that pulmonary stenosis furnishes a very decided predisposition to phthisical changes in the lungs, and Leyden showed that the abnormally small amount of blood received by the lungs favors the drying and caseation of inflammatory products (probably also the infection with bacilli) in the respiratory passages. Phthisical processes in the lungs may also develop at times in other valvular lesions. Frommolt found it twenty-two times in two hundred and seventy-seven autopsies upon cases of valvular disease. He states that phthisis occurs with greater relative frequency in diseases of the aortic than of the mitral orifice, and Traube long since attributed this fact to the more profuse impregnation of the pulmonary parenchyma with blood serum under the last-mentioned circumstances.

The simultaneous affection of several cardiac orifices is said to be hardly ever associated with phthisis, unless the pulmonary orifice is stenosed. Nevertheless, the effect of valvular lesions on the development of phthisis cannot be disputed. For, although valvular lesions may develop in individuals suffering from pulmonary phthisis, the latter disease rarely begins in those who are already suffering from valvular lesions (with the exception of pulmonary stenosis), even if an hereditary predisposition is present.



16. *Course and Termination.*—We have previously mentioned that valvular lesions may recover clinically, but this occurs so exceptionally that it cannot be taken into consideration in prognosis. Jaksch, however, regards this termination as more frequent, and believes that dilatation of the intact leaflets may overcome the defects of the diseased ones, although this often requires years. The observations of Jaksch at the post-mortem table do not agree with the results of clinical observation.

The duration of the disease depends on the nature of the lesion and the strength of the patient. As a rule, aortic lesions are compatible with a longer lease of life than other valvular lesions. Fothergill recently reported a case of aortic insufficiency of twenty-five years' duration. This circumstance is owing to the fact that the left ventricle, on account of its thicker muscular walls, is better able to fulfil the increased demand for work than the right ventricle.

If a valvular lesion develops in early childhood, the disease usually runs a shorter course, because the growth of the body requires a lively demand upon the supply of vital energy. Death may occur in a few weeks or months in feeble individuals in whom the development of compensation is rendered difficult or impossible.

The fatal termination generally follows increasing disturbance of compensation, the result of fatty degeneration of the heart muscle. Death occurs usually from edema of the lungs, paralysis of the heart, or exhaustion.

In other cases death occurs unexpectedly. Thus patients have been known to drop dead immediately after a long walk or railroad trip, or after dancing. Death may also result from rupture of the heart, cerebral hemorrhage or embolism. Finally, intercurrent diseases may prove fatal, either per se or by giving rise to secondary changes in the heart muscle.

III. ANATOMICAL CHANGES.—With regard to the changes in the heart, it must be remembered that not every thickening or tissue change in the valvular apparatus is associated with a disturbance of function. Hence, the anatomico-pathologists consider valvular lesions much more frequent than is conceded by clinicians. On the other hand, there are cases in which the valvular apparatus is intact, but in which the orifice is excessively dilated, or it is narrowed by tumors or clots, or the unfolding of the valves is obstructed mechanically by tumors in the neighborhood, or the number of leaflets is congenitally greater or less than normal. In such cases, observation during life is often more positive than the anatomical demonstration of the valvular lesion.

To demonstrate insufficiency of the semilunar valves, we should remove the heart as high as possible on the main arterial trunks, cut the latter a little lengthwise, draw them apart like a funnel at the upper cut ends, and allow a vigorous stream of water to fall into the aorta from a moderate height. The semilunar valves will then unfold, become applied to one another and permit a very slow escape of the water. But if the valves are insufficient, the water will at once run through.

The anatomical demonstration of stenosis of the arterial orifices is not difficult, as a rule, because the changes are so palpable. With regard to the venous orifices it must be remembered that they readily admit the passage of the index and middle fingers under normal conditions.

Numerous measurements of the various orifices have been made.

But great individual variations are possible, and in doubtful cases the figures do not help us much more than the coarser methods of examination mentioned above. Luschka gives the following figures:

Circumference of the aortic and pulmonary orifices,	7	cm.
“ “ left auriculo-ventricular orifice,	8.5	“
“ “ right “ “ “	10.	“

Most authors agree, however, that the pulmonary orifice is somewhat larger than the aortic orifice; according to Peacock, the difference is a little over 1 cm.

Valvular insufficiency may be produced by the following anatomical changes in the tissue of the valves:

a. Rupture of the valves from the chordæ tendinæ or points of insertion. More rarely a rent passes directly from the free border of the valve through the leaflet.

b. Aneurism of the valve and perforation of the valves.

c. Adhesion of the valves to the wall of the ventricle or the wall of the aorta or pulmonary artery, and also adhesion of the valves and chordæ tendinæ.

d. Thickening of the valves, followed usually by retraction of the tissue, thickening and retraction of the chordæ tendinæ, and fibroid degeneration of the papillary muscles.

e. Endocarditic vegetations of a certain size may also cause insufficiency of the valves.

In stenosis of the valvular orifices we find:

a. Endocarditic vegetations on the valves.

b. Adhesion of the valves to one another.

c. Thickening and calcification of the valves, so that they are no longer pushed aside by the current of blood.

d. Processes of retraction at the ring of insertion or at the free border of the valves.

As in cases of insufficiency, several of these factors are usually combined, and the stenosis may become so extreme that it is hardly possible to pass the tip of a sound through the narrow slit between the valves.

The parietal endocardium very often shows opacities and thickenings (tendon patches of the endocardium). Attention has been previously directed to the secondary changes in the heart muscle, and to the fact that pericardial affections are found not infrequently.

We will not be able to give a full anatomical description of the other organs, but must be satisfied with briefly mentioning the manifold changes.

If symptoms of stasis have been present, the veins are markedly distended. The blood is usually blackish-red, thin, and in some cases contains an abnormally small amount of solid constituents, particularly of albumin (Becquerel and Rodier).

The pericardial, pleural, and peritoneal cavities often contain considerable amounts of a clear, amber-yellow fluid. This has a bloody color occasionally, and particularly if signs of blood dissolution have been present during life. Cloudy fluid is observed in rare cases, in other words, an exudative process has been present either from the start, or has been superadded to the original transudative process.

Bronchial catarrh, œdema of the lungs or ary-epiglottic folds (œdema of the glottis), and pneumonic changes may be observed in the air passages. The anterior median borders of the lungs are often emphysematous. The lungs are not infrequently pigmented with a peculiar brown red, and are denser than normal; the microscope reveals increase of the interstitial connective tissue and a large amount of blood pigment, the capillaries of the alveoli are very sinuous, and in part extend far into the lumen of the pulmonary alveoli. The blood pigment is situated in part in the endothelium of the alveoli, in part it is free in the alveoli,

and in part it is situated in the interstitial connective tissue. In a case described by Orth, the capillaries and even larger vessels were filled with masses of pigment. These changes are known as brown induration or pigment-induration of the lungs. Fresh extravasations of blood are sometimes found in the lungs. Buhl showed that the small vessels of the lungs undergo fatty degeneration not infrequently as the result of valvular lesions, and this process may be associated with the frequent occurrence of hemorrhagic infarctions in heart disease. These must be distinguished from the hemorrhages following embolism; the latter are recognized by their wedge shape (wedge-shaped infarctions). The broad base is directed towards the periphery, the apex towards the hilus of the lungs.

The conditions just described are sometimes followed by gangrenous processes, which may extend to the pleura, perforate it, and give rise to pneumothorax.

The laryngeal tissues sometimes present cedematous swelling, but this may have been present during life without being demonstrable after death.

The spleen is not infrequently enlarged to a slight extent. Its capsule is often thickened, and is sometimes adherent to adjacent organs. Upon section, the tissue of the spleen is found to be firm, the result of proliferation of the trabecular connective tissue. The organ not very infrequently contains wedge-shaped infarctions which vary considerably in shape according to their age and termination.

There is very often catarrhal swelling of the mucous membrane of the stomach and intestines. Hemorrhagic erosions or diffuse bloody infiltrations and bloody gastro-intestinal contents are also observed at times. The hemorrhoidal veins are found dilated.

Very important changes appear in the liver. The organ is often strikingly large and congested; it is firm and blackish-red in color. Upon section, the central veins of the acini are found filled and dilated, giving rise to branching reddish figures, which have been compared to the appearance of an oak leaf or a nutmeg blossom (cyanotic nutmeg liver). On microscopical examination, the central veins and intralobular blood-vessels are found greatly distended, and may have given rise to atrophy of some of the hepatic cells. In addition, there are not infrequently fatty changes in the cells, so that a very distinct contrast is produced between the gray, fatty peripheral zone of the acini and their hyperemic centre.

If the stasis has existed for a long time, the dilatation of the intralobular vessels is followed by proliferation of the connective tissue which afterwards undergoes retraction. The size of the liver is thereby diminished, its consistence becomes remarkably firm, and the surface is uneven and nodular. The centre of the acini, which is filled with blood, remains very distinct (atrophic nutmeg liver). These changes must not be mistaken for cirrhosis of the liver.

Catarrhal swelling and extravasations of blood are observed upon the mucous membrane of the gall bladder. The bile is generally very thin and watery.

The kidneys are often found to be marked swollen, increased in size, and congested. The capsule is very tense, but usually transparent, and easily detached from the parenchyma. Upon the surface of the kidney, the scars of Verheyen are distended, and readily traced with the naked eye into their finest ramifications. Upon section, the difference between cortex and pyramid is very distinct, the latter being blackish-red or bluish-red. The bluish-red color is more marked at the basal portion. Under the microscope, the Malpighian bodies and tunica propria of the tubules are slightly thickened, and often present a distinctly streaked appearance. The epithelium of the tubes is generally in a condition of cloudy swelling, sometimes fatty and desquamated. Granules and needles of blood pigment may be found in the tubes. The fatty degeneration of the epithelium increases after the congestion of the kidneys has lasted a long time. A uniform proliferation of the interstitial connective tissue generally occurs then in all parts of the kidneys, and when this undergoes retraction at a later period, the increase in size disappears, the surface becomes granular, and the condition may finally be mistaken for true cirrhosis of the kidney. The organ sometimes contains wedge-shaped infarctions in various stages. We have already mentioned that valvular lesions may develop during the course of true nephritis.

Catarrhal swelling and hemorrhages may be noticed on the mucous membrane of the urinary passages.

Chronic congestion of the uterus is very often observed.

The pancreas may be changed by venous congestion and hemorrhages.



Fatty degeneration and atheroma of the intima of the aorta are found not infrequently.

Very important changes occur in the central nervous system. The inter-meningeal spaces and cerebral ventricles often contain an excessive amount of serous fluid. An accumulation of fluid also occurs in the tissue of the pia mater. The meninges are not infrequently thickened and opaque; thickening of the ependyma is also noticed. The vessels of the pia mater and the sinuses contain a large amount of blood. Finally, meningeal and cerebral hemorrhages may be present.

The embolic aneurisms described by Ponfick are observed occasionally on arteries situated in a yielding parenchyma. They are produced in the following manner: A calcified portion of a valve is carried off by the current of blood, and remains in an artery without obstructing it completely. Its sharp edges gradually perforate the walls of the vessel, and if the consequent extravasation of blood takes place gradually, and is not too large, a false aneurism is produced. If these aneurisms burst, they may give rise to large hemorrhages.

IV. DIAGNOSIS.—The diagnosis of the individual valvular lesions depends exclusively on the results of physical examination of the heart, for which we refer to preceding sections. At the present time, we will discuss merely the difficulties in the way of diagnosis.

As a general thing, those valvular lesions which give rise to a diastolic murmur are diagnosed more readily than those producing a systolic murmur, *i. e.*, insufficiency of the semilunar valves and stenosis of the auriculo-ventricular valves are recognized with greater certainty than stenosis of the semilunar valves and insufficiency of the auriculo-ventricular valves. This is owing to the fact that diastolic murmurs are almost always the result of valvular lesions, while systolic murmurs are observed as accidental murmurs in anæmic and febrile conditions. A valvular lesion can be diagnosed from the presence of a systolic murmur only when hypertrophy and dilatation of a part of the heart can also be demonstrated. A systolic murmur and dilatation of a part of the heart in themselves are insufficient, as this occurs not infrequently in febrile and anæmic conditions and pulmonary emphysema, although a valvular lesion is not present. But in the discussion of cardiac dilatation and hypertrophy, we called attention to the fact that certain conditions may conceal a change in the heart-muscle and others may simulate it, so that it is evident that difficulties in diagnosis may arise. As a rule, it may be held that accidental murmurs do not give rise to thrill, though exceptions sometimes occur.

Errors in diagnosis may also arise when the rapidity of the blood current is slight and endocardial murmurs are not present at all times. An individual may then be regarded as healthy, although the valvular apparatus is seriously affected. These diagnostic dangers will be avoided by him who reserves a diagnosis until the heart has been examined after the rapidity of the circulation is artificially stimulated. For this purpose, the patient should walk rapidly about the room, bend down and raise himself a number of times in rapid succession, take deep and rapid respirations; he should be ausculted in the recumbent, sitting, and standing positions during inspiration and expiration.

Concerning the localization of endocardial murmurs, Skoda's rule holds good, *viz.*, the murmur is produced at that valve over which it is heard with greatest intensity. But there are exceptions to this rule. Thus we have shown that in aortic insufficiency the diastolic murmur is often louder over the middle of the sternum than over the artery, and even in mitral insufficiency the systolic murmur may be loudest over the pulmonary orifice.

Murmurs over the pulmonary orifice may sometimes be mistaken for

aortic murmurs, those over the tricuspid for mitral murmurs. To prevent mistakes, we should direct our attention to the dilatation and hypertrophy of individual parts of the heart, to other symptoms (*pulsus durus et celer* in aortic insufficiency, venous and hepatic pulse in tricuspid insufficiency), and should also bear in mind the rule laid down by Oppolzer, that pulmonary and mitral murmurs persist as such when the stethoscope is moved to the left of the heart, while pure sounds become audible if the murmurs are conveyed. This is also true, but in an opposite direction, of the aorta and tricuspid.

The diagnosis may be very difficult in a combination of lesions at a number of valves. Attention should then be paid specially to the timbre of the murmurs. For if synchronous murmurs of different timbre are heard over several valves, we may justly assume that we have to deal, not with conveyed, but with different autochthonous murmurs.

V. PROGNOSIS.—The prognosis of valvular lesions of the heart is unfavorable. Spontaneous recovery occurs occasionally, but it is exceptional and nothing can be effected by medication. The opportunity for dangerous intercurrent attacks is very great, and if these dangers are fortunately avoided, the time arrives when the heart muscle becomes insufficient and symptoms of increasing disturbance of compensation lead to death. In the majority of cases this is the result of fatty degeneration of the heart.

The nature of the valvular lesion must also be taken into consideration in prognosis. Aortic lesions are usually more favorable than other forms, because the left ventricle, with its extensive muscular walls, is more adapted to compensatory changes. Lesions of the pulmonary valves are especially unfavorable, because a fatal termination from pulmonary phthisis is impending, in addition to the dangers arising from the valvular lesion.

The rule also holds good that combined lesions of several valves are more unfavorable than disease of a single valvular apparatus. This is owing to the fact that an especially great amount of labor thereby devolves upon the heart.

The social position of the patient possesses a significance in prognosis which should not be underestimated. The greater the amount of bodily rest which can be secured, the greater the chance of delaying disturbances of compensation or relieving those already existing.

Constitution and age also exert some influence. The more vigorous the constitution, the greater the hope that the heart may fulfil the increased requirements demanded of it. If valvular lesions develop in early childhood, there is danger of early disturbance of compensation and a rapid fatal termination. An especially grave prognostic significance has been attributed to certain symptoms. We refer particularly to *cardialgia*. Repeated attacks of palpitation, increasing want of breath, and irregularity of the heart's action (*delirium cordis*) intermittence of the pulse, in which not every systole is sufficient to drive the blood to a perceptible extent into the radial artery, and the ventricle is often not entirely emptied during systole (*asystole* of Beau), increasing feebleness of accentuation of some of the heart sounds, for example, the second pulmonary sound in mitral lesions, increasing dilatation of the heart, diminished strength and diffuse character of the apex beat, are of evil significance, as they indicate the threatening or existing disturbance of compensation.

VI. TREATMENT.—The therapeutic indications are the cure of the

valvular lesion, the prevention of disturbances of compensation, and the removal of those disturbances when present.

The cure of a valvular lesion by medication can hardly be expected. It was formerly believed that the protracted use of potassium iodide would cause disappearance of the inflammatory changes in the endocardium, but this has not been confirmed. This is also true of treatment with mercurials and repeated blood-letting. Gerhardt reports considerable improvement, even in chronic endocarditis, from inhalations of sodium carbonicum (1-1.5% two to four times a day for one-quarter of an hour). English writers ascribe a solvent and absorbent effect to ammonium chloride. Johnson vaunts the effects of repeated blisters, applied directly over the affected orifices. The iodide treatment is generally hurtful, but the other remedies mentioned may be employed for want of something better.

Beneke was the first to recommend cool-baths for the absorption of fresh endocarditic vegetations.

In the majority of cases, our object is to maintain compensation unchanged as long as possible. This is done with greatest certainty by dietetic measures, and it is a rule that no drugs should be administered except in cases of necessity.

The patients must avoid all bodily and mental fatigue. Gymnastics, dancing, riding, running, mountaineering are tabooed. Long pedestrian trips may become very dangerous, and this is also true of long journeys on the railway. Nevertheless, the patients should exercise daily in the open air, but not sufficiently to give rise to palpitation, dyspnoea, and excessive exhaustion. Special caution is necessary in the use of cold baths. River and sea baths should only be taken, if at all, in the presence of others. I have seen, on a number of occasions, that patients with heart disease were seized in a cold bath with sudden attacks of anxiety, palpitation, and pain in the heart, and ran the risk of suffocation. The effect of cold water on the cutaneous vessels, and thus on the entire circulation, may be attended with very grave consequences. Care should also be exercised in the use of warm baths. Cold frictions may be recommended when they are not followed by any bad symptoms.

Articles of food which are digested with difficulty and have a tendency to bloat should be avoided. Milk, eggs, soup, lean meats, cooked fruit, and the like may be particularly recommended. The meals should be frequent, but the quantity ingested not too large, as a heavy meal is apt to produce palpitation, dyspnoea, and anxiety. A moderate amount of beer and light white wines may be permitted, and should be recommended if the nutrition of the patient is impaired. Strong coffee, tea, and liquors are generally injurious and should be discontinued as soon as they produce any disturbances on the part of the heart.

The bowels should be kept open daily, either with the aid of cooked fruit or, if necessary, of mild laxatives, for example: Aloes, Ext. rhei,  $\text{āā}$  1.5. Pulv. et succ. liquirit., q. s. ut ft. pil. No. 30. D. S. two to four pills every night; Aloes, Ext. rhei, Tub. jalap.,  $\text{āā}$  1.0. Pulv. et succ. liquir., q. s. ut ft. pil. No. 30. D. S. two to four pills every night. Bitter waters (Friedrichshall, Puellna, Saischuetzer, Hunyadi János, a wineglassful before breakfast, followed by a glass of drinking water), and laxative mineral waters (Marienbad, Homburg, Kissingen, Seidlitz, etc.) are often indicated. Grape and whey cures act in the same way.



A frequent change of residence is often very desirable. The patients often feel particularly well in a mountainous region, but the locality should contain level walks. Great caution should be exercised in visiting the sea shore, because the subjective complaints of many patients are thereby increased. A Southern climate may be advantageous during the cold months.

When signs of disturbed compensation appear, the treatment varies according as we have to deal with temporary conditions of heart weakness or fatty degeneration of the heart muscle.

Among the remedies for increasing the vigor of the heart's action, the greatest reliance should be placed on digitalis and its preparations. It is particularly serviceable in delirium cordis and asystoly, because it possesses a decided regulating influence in addition to its tonic effect. We may give *infus. fol. digitalis*, 0.5–2.0 (200.0), to which *tartarus depuratus*, 10.0, may be added if there is a tendency to constipation, or *kali nitricum*, 10.0, or *liq. kali acetic*, 30.0, if diuresis is scanty. If diuresis is very much diminished, the following prescription may be recommended: *Kali carbonic.*, 5.0; *succi citri*, q. s. ad sat., *inf. fol. digital.*, 0.5–2.0 : 150. M. D. S. One tablespoonful every two hours. The effect of the digitalis should be carefully watched every day, as it must generally be discontinued after the second or third bottle. The symptoms of poisoning are great slowness of the pulse, irregularity of the pulse, feeling of confusion in the head, spots before the eyes, ringing in the ears, vomiting, pain in the epigastrium, diarrhœa, and general symptoms of collapse.

Caffeine, *adonis vernalis*, *convallaria majalis* have been recommended recently, in addition to digitalis, to strengthen and regulate the heart's action, particularly after symptoms of digitalis poisoning have set in.

We have been very well pleased with the action of *cafeinum citrico-benzoicum* (*aq. dest.*, *glycerin.*,  $\text{ââ}$  5.0; one syringe-ful subcutaneously once or twice a day). It slows, regulates, and strengthens the heart's action, and particularly increases diuresis. It may be given for weeks without special unpleasant incidental effects, but the heart soon becomes accustomed to it, and it then loses its effect. Some patients complained of violent pains, occasionally lasting an hour, at the site of injection.

*Adonis vernalis* (*inf. adon. vern.*, 5.0 : 150.0. One tablespoonful every two hours) has a similar though less certain effect; a few patients complained of nausea and pain in the abdomen.

*Convallaria majalis* we regard, to say the least, as a very unreliable preparation. It has been recommended as infusion 5–10 : 150, one tablespoonful every two hours; as tincture, 20 drops t. i. d.; as aqueous extract, 5–12 drops four times a day.

Clifford Allbutt has recommended tincture or infusion of *prunus virginianus* as a substitute for digitalis.

A tonic action on the heart muscle has been justly ascribed to small doses of quinine. Papillaud also recommended arsenic and antimony preparations, particularly *stibium arsenicosum* in pill form 0.001, twice a day. Cheral prefers *ammonia arseniate*, 0.01, twice a day.

In anæmic individuals, preparations of iron are advisable, associated with digitalis and quinine (*ferri hydrogenio reduct.*, 10.0; *fol. digital. pulv.*, *chinin. muriat.*,  $\text{ââ}$ , 2.0; *pulv. althææ*, q. s. ut ft. pil. No. 50. D. S. One pill four times a day after eating). Ferruginous waters may also be serviceable, particularly those containing Glauber's salts or chlo-

ride of sodium. Scholz maintains that iron baths containing carbonic acid of an indifferent temperature often act better than digitalis.

If we have reason to suspect fatty degeneration of the heart muscle as the cause of the disturbance of compensation, little can be expected from digitalis. In such cases the treatment should be more stimulating. We may allow tea, the stronger kinds of wine (champagne is often indicated), ether, camphor, castoreum, etc.

The constant application of an ice-bag to the præcordial region is indicated if the patient suffers a great deal from palpitation. If the palpitation is constant, advantage may be derived from wearing over the heart lead flasks filled with cold water. If the palpitation is associated with great anxiety and painful sensations around the heart, subcutaneous injections of morphine (morphin. muriat., 1.0; glycerin. puri, aq. dest., ãã 15.0. M. D. S. One-half syringeful) sometimes exert a magical effect. But this remedy should be looked upon as a *dernier ressort*. The employment of narcotics in heart disease is not devoid of danger, and, in addition, the dose must soon be increased. If there is fatty degeneration of the heart muscle, injections of morphine may give rise to serious disturbances in the innervation of the respiratory muscles (Cheyne-Stokes respiration, vide page 57).

Pulmonary symptoms may require special treatment. Expectorants are indicated in bronchial catarrh, excitants in œdema and inflammation of the lung, styptics in hemorrhagic infarctions.

Compressed and, also, rarefied air have been repeatedly employed with good effects. In aortic insufficiency, Fenoglio has recommended expiration in rarefied air. We will mention, finally, that douches and cold frictions, and even the Swedish movement cure, have been recommended as specifics against valvular lesions.

### 3. Congenital Valvular Lesions.

#### (*Cyanosis congenita s. Morbus cœruleus.*)

I. ETIOLOGY.—Valvular lesions may develop during fœtal life, but they are rare in comparison with acquired lesions. Pulmonary stenosis is relatively most frequent, so that in cases of congenital valvular lesion there is a certain amount of justification for suspecting this form of disease.

It is generally held that acquired lesions affect almost exclusively the valves on the left side of the heart; congenital lesions those on the right side, but exceptions to this rule are sometimes observed.

Congenital lesions are more frequent in male than in female children. Hereditary influences are noticeable in some cases, and a number of families have been described in whom congenital valvular lesions occurred during several successive generations. I am acquainted with a case in which a child by the man's first wife, and one by his second wife, suffered from congenital valvular lesion of the heart.

In the majority of cases no cause can be ascertained. Injuries and mental excitement during pregnancy have been mentioned as causes.

Congenital lesions of the heart affect not only the valves and orifices, but also the ventricular and auricular septa and the ductus Botalli. As a rule, a single valvular apparatus is rarely affected alone, but there are almost always a number of lesions.

At one time almost all congenital valvular lesions were attributed to

fœtal endocarditis, but the view has gained ground recently that they are the result of inhibited development. It is well known that numerous changes occur during the embryonal development of the heart, and it is not astonishing, therefore, that its development is readily disturbed and inhibited.

There can be no doubt that endocarditic changes are found with extreme frequency at the autopsy, but v. Dusch has shown that these may have developed at a later period, and that the patency of fœtal channels of circulation predisposes to inflammatory changes at the sites of abnormal communication. At all events, it is evidently difficult or impossible to decide, from an anatomical standpoint, whether the congenital valvular lesion should be regarded as the result of inflammation or inhibited development. But it is readily understood that, if errors of development occur at any part of the heart, other parts are also readily implicated, because the one process passes directly into the other. As a matter of course, however, we do not maintain that congenital valvular lesions never result from fœtal endocarditis.

The greater frequency of congenital lesions on the right side of the heart has been attributed to the fact that it performs the greatest amount of work during fœtal life, and contains the blood which has been arterIALIZED in the placenta, so that it assumes to a certain extent the later functions of the left heart.

**II. ANATOMICAL CHANGES.**—We will here discuss only those lesions which possess a clinical interest.

*a.* Among congenital lesions of the heart, stenosis or atresia of the pulmonary orifice has always attracted the chief attention. The changes are not always confined to the orifice proper, but may affect the conus arteriosus, pulmonary orifice, and the trunk of the pulmonary artery.

Stenosis and atresia of the conus arteriosus occur at the ventricular origin or immediately beneath the pulmonary orifice, or they affect the entire conus uniformly. In the first event, the conus is not infrequently separated from the right ventricle and converted into an independent cavity—the third or supernumerary ventricle. Changes at the conus are often associated with malformations of the pulmonary valves; they are not developed regularly or are abnormal in number or present endocarditic changes. In many cases the stenosis is formed by a ring-shaped, connective-tissue callosity, so that it looks, at first sight, like a myo-endocarditic process. In other cases muscular ridges project from the inner wall of the conus and narrow its lumen.

Changes in the pulmonary valves are never absent in stenosis of the pulmonary orifice itself. Endocarditic proliferations, calcification, and adhesions of the valves are very often noticed. Occasionally all the leaflets have united into a sort of diaphragm which is stretched across the origin of the pulmonary artery. Thickening and retraction of the valvular ring are also observed.

Atresia and stenosis of the trunk of the pulmonary artery are associated generally with similar changes at the pulmonary orifice. In the most marked cases the pulmonary artery is converted into a solid cord, which terminates in a blind extremity at the pulmonary orifice. Sometimes the change is continued into a part of one or the other main branches of the pulmonary artery.

The conditions referred to are associated very generally with abnormalities of the septa of the heart and of the fœtal circulatory channels.

If the pulmonary stenosis develops before the end of the second fœtal



month, *i. e.*, at a time when the upper part of the septum ventriculorum is not yet closed, the latter remains patent and thus renders possible the passage of blood from the right into the left ventricle. At the same time the auricular septum is not infrequently situated very far to the left, so that the aortic orifice opens partly above the right, partly above the left ventricle, or takes its origin altogether from the right ventricle. In some cases the ventricular septum is developed so slightly that it is merely indicated by a narrow projection.

If the pulmonary stenosis develops after the closure of the ventricular septum, the passage of blood from the right into the left ventricle is only possible if there is an opening in the auricular septum. Patency of the foramen ovale is most frequent; more rarely there are openings in the septum proper. At the openings in the septa we often find endocarditic proliferations, callous induration or calcification, indeed acute endocarditis may develop there, extend to the valves and, in later years, add an acquired lesion to the congenital one.

Openings in the septa are not always present in pulmonary stenosis: when present, they are almost always associated with patency of the ductus Botalli. This then assumes the office of allowing the passage of blood from the aorta into the main branches of the pulmonary artery and of compensating in this way for the diminished supply of blood to the lungs. As a matter of course the current of blood flows in an opposite direction to the normal fœtal current, since the ductus Botalli, under normal conditions, conveys the blood from the pulmonary artery into the descending aorta.

But the ductus Botalli may be closed or entirely wanting, and life may be maintained even if this condition is associated with complete obliteration of the pulmonary artery. Under such circumstances the supply of blood to the lungs must be conveyed by branches of the aorta, and the bronchial, œsophageal, and pericardial arteries assume this function. Weiss described a case in which an anomalous artery, which was given off from the aorta immediately above the diaphragm, furnished a direct connection between the aorta and pulmonary artery.

If we bear in mind that these changes may be combined in a manifold manner, we will readily understand that the anatomical appearances in congenital pulmonary stenosis may present a very variable picture.

The right ventricle is generally in a condition of hypertrophy, or if, as is frequent, pulmonary insufficiency is present in addition to the stenosis, the right ventricle also undergoes dilatation. On the other hand, the right ventricle is atrophic if the pulmonary orifice is entirely obliterated and the ventricular septum is closed. There are often malformations of other valves and orifices, together with abnormal origin of the large vessels.

*b.* The congenital defects of the auricular septum are patency of the foramen ovale and openings in the tissue of the septum proper.

Patency of the foramen ovale is very frequent. Wallman and Klob found it 356 times in 800 autopsies (44.5%). At the anterior end of the oval opening is usually found a slit-shaped opening which admits the passage of a sound from the right into the left auricle. Abnormalities of the valve and the oval opening may be entirely absent. In other cases the valve membrane of the foramen ovale is perforated, either like a sieve or in the shape of several slit-like openings. In other cases finally the valve membrane is developed too imperfectly to close the foramen ovale, or the latter is so large that a membrane of normal size is incapable of

closing it. The opening is sometimes so extensive that it readily admits the passage of a finger.

Patency of the foramen ovale may be expected when the conditions of blood pressure are disturbed after birth. During the foetal period the foramen is open so that the blood flows in part from the right auricle into the left. Immediately after birth the oval opening is closed because the blood pressure in the left auricle equals that in the right, so that the valve of the foramen is closely applied to its wall. In the first weeks of life the anterior free edge of the valve becomes adherent under normal conditions, but this adhesion remains absent or is incomplete if the pressure in the left auricle is abnormally low. This occurs if the flow of blood to the left auricle is diminished during the first days of life on account of pulmonary atelectasis or pneumonia. In other cases we find openings in the auricular septum associated with congenital diseases of the valves and orifices. These include occlusion or stenosis of the pulmonary and aortic orifices or the auriculo-ventricular orifices, transposition of the large vessels, the presence of only one ventricle. Under such circumstances the circulation is only rendered possible or at least aided by the existence of the openings referred to. In a last group of cases there are primary defects in the septum without circulatory disturbances.

Openings in the primary ventricular septum are much rarer. There may be only one or several, and indeed may be present to such an extent that the auricular septum is entirely absent. They result from the fact that the auricular septum in its development does not extend to the ventricular septum.

c. Defects of the septum ventriculorum may be single or multiple and in some cases the septum is barely indicated by a narrow prominence. They are sometimes found in an otherwise well-developed heart. In other cases there are congenital adhesions or stenoses at the venous or arterial orifices, so that circulation is only rendered possible by the defects in the septum. In a third series of cases we find myocarditic callosities, induration, calcification or endocarditic proliferations upon the edges of the opening, giving rise to the impression that the defect is the result of inflammatory processes.

Defects in the ventricular septum are sometimes associated with others in the auricular septum. Most frequently there is an opening which begins under the right leaflet of the aortic valve and passes into the lumen of the conus of the pulmonary artery. But the opening may also be situated in such a position as to connect a ventricle with the auricle of the opposite side, or to make a communication between all four cavities of the heart. Cruveilhier and Heschl described cases in which a narrow canal started from the aortic valve and emptied into the right ventricle near the apex.

d. Among congenital diseases at the right auriculo-ventricular orifice may be mentioned: stenosis and atresia of the orifice, and insufficiency of the tricuspid valves.

Stenosis and atresia of the orifice may be the result of foetal endocarditis or of abnormal development. If the orifice is entirely closed, there is always an opening in the ventricular septum, for in this way alone can the right ventricle, and from it the pulmonary artery, be supplied with blood. At the same time there are openings in the foramen ovale, so that the blood may pass from the right auricle into the left heart. But if the lumen of the pulmonary artery is also occluded, the lungs can only be supplied with blood by regurgitation from the aorta through the

ductus Botalli into the branches of the pulmonary artery. Atresia of the auriculo-ventricular orifice is followed by atrophy of the right ventricle which is especially marked when stenosis or atresia of the pulmonary artery is also present.

*e.* Congenital diseases of the ductus arteriosus Botalli include its absence, premature closure, and patency of the canal.

Under normal conditions the ductus Botalli is closed soon after birth. This is rendered possible by the fact that, after the beginning of respiration and expansion of the lungs, the blood pressure in the pulmonary artery falls, so that the passage of blood from the pulmonary artery into the aorta no longer occurs. The closure is effected by proliferation of the layers of the intima and media; this begins at the middle of the canal, spreading first towards the pulmonary, then towards the aortic opening. At the end of the third week the canal is no longer permeable, and at the end of the fourth week the obliteration is complete.

Premature foetal occlusion is sometimes observed in atresia of the pulmonary orifice, because the ductus receives so little blood from the pulmonary artery that obliteration may take place.

Patency of the ductus Botalli is sometimes the result of mechanical causes, sometimes of congenital anomalies of the wall of the vessel, which seems to possess but little tendency to proliferating and obliterating processes. Rokitansky demonstrated a distinct arterial structure in the patent ductus Botalli, and according to Gerhardt the patent canal is not infrequently unusually long. We have previously referred to mechanical causes of its patency, since it is observed in various congenital heart lesions, if the necessity arises for the pulmonary artery to receive blood from the aorta or vice versa. Atelectasis and pneumonia during the first few days of life sometimes appear to favor the patency of the canal, inasmuch as they aid the continuance of the foetal flow of blood from the pulmonary artery into the aorta.

The shape of the ductus is not infrequently changed. In some cases it presents aneurismal dilatations, or the aortic end may be dilated and funnel shaped. In rarer cases the ductus is so short that the aorta and pulmonary artery appear to be in direct communication with one another by means of an opening.

*f.* Congenital stenosis and atresia of the aortic orifice are not frequent. They are the result of foetal endocarditis and myocarditis or of defective development, and anomalous division of the truncus arteriosus communis. In the first case, according to Rauchfuss, the ventricular septum is closed, but the foramen ovale is necessarily open to convey part of the blood from the left into the right heart. The inflammation must have occurred prior to the third foetal month. If the lesion is the result of disturbed development, the ventricular septum is found open; atresia, rather than stenosis of the aortic orifice is generally found in such cases. Stenosis of the mitral orifice and malformation or inflammation of the mitral valve are very generally present at the same time.

In atresia of the aortic orifice the left ventricle undergoes atrophy; this is particularly well marked if the mitral orifice is also stenosed. Circulation in the aortic system is hardly possible unless the descending aorta and from it the arch of the aorta (by regurgitation) receive blood from the pulmonary artery through the agency of the ductus Botalli. The right heart, particularly the auricle, is increased in size, as it receives an unusually large amount of blood.

*g.* Congenital stenosis and atresia of the mitral orifice are rare. They



may be associated with mitral insufficiency; in addition, there are usually openings in the ventricular and auricular septa.

*h.* Transposition of the cardiac arterial trunks is a term applied to that condition in which the aorta takes its origin from the right, and the pulmonary artery from the left ventricle. The foramen ovale is patent, because through it alone can the blood flow from the right into the left auricle and be partially arterialized in the lungs. There are often, in addition, defects in the ventricular septum. The circulation is not disturbed much if the veins are also transposed.

Sometimes the venous trunks alone are transposed. In this event, also, circulation can only be carried on if the foramen ovale remains open.

III. SYMPTOMS AND DIAGNOSIS.—The symptoms of congenital valvular lesions are divided into general and local. Among the former, cyanosis is a prominent symptom, but is not observed constantly. While it attracts attention in some cases at birth, in others it does not appear until after years, or it may occur only at times, after physical or mental excitement or when the patients have been exposed to cold air.

The cyanosis usually is extremely well marked so that the the skin looks as if painted blue. The small cutaneous vessels rather than the large veins are implicated in the cyanosis.

Children suffering from congenital pulmonary stenosis are very often asphyxiated, and of a bluish-red color at birth. If respiration is begun, the cyanosis disappears to a certain extent, and becomes more marked only when the child cries. The cyanosis may also disappear during serious diseases which are associated with anaemia. The children have a tendency to convulsive seizures; attacks of asthma and suffocation are also observed, and sometimes are the immediate cause of death.

The cyanosis is the result of stasis and slowness of circulation in the domain of the *venae cavae*. The view that it is caused by admixture of the venous blood of the right ventricle with the arterial blood of the left heart, on account of openings in the septa of the heart, is contradicted by the fact that cyanosis is sometimes absent in cases in which the ventricular septum is entirely wanting, but has been observed in individuals in whom these openings were not present. Moreover, Brechet observed a case in which cyanosis was absent, although the left subclavian artery took its origin from the pulmonary artery, so that the left arm received only venous blood.

As a rule, the radial pulse is not full; in some cases the left pulse was weaker than the right. The bodily development is generally impaired. The patients are feeble, lean, and have poorly-developed muscles. The genitals are also poorly developed. The mental development is impaired, and the patients are quiet and apathetic. The eyeballs are occasionally prominent. The bluish lips and the nose are often very prominent, according to Foerster, on account of serous transudations.

Bouchut and Gatti found the optic papillæ congested, the retinal veins dilated and sinuous, the arteries and veins were alike red.

The tips of the fingers are club-shaped, and the fingers are sometimes claw-shaped. The cyanosis is remarkably intense under the nails, the latter are often long, hard, and thickened. There is sometimes a great tendency to panaritias.

The patients often complain of severe chilly sensations. The skin is cool, and the axillary temperature abnormally low. Further investigation

is necessary to determine whether this corresponds to low rectal temperature.

Many patients present a marked tendency to hemorrhage, either from the nose, gums, or the bronchial passages and lungs. There is a special predisposition to chronic cheesy processes in the lungs, so that the majority of the patients die of pulmonary phthisis. The pulmonary changes generally begin on the left side, and remain more marked there than on the right side. Symptoms of stasis and œdema, to which the patients succumb, develop more rarely.

The outward appearance of congenital cyanosis is so striking that the suspicion of heart disease is aroused at the first glance. The character of the latter can only be determined, if at all, from the local changes in the heart, and these alone can be taken into consideration in cases in which the cyanosis is absent. But the local changes themselves are not infrequently of such a doubtful character that the autopsy alone reveals the congenital heart lesion.

*a.* Congenital stenosis or atresia of the *conus arteriæ pulmonalis*, the pulmonary orifice or artery, gives rise to the symptoms described on page 101. We will find, therefore, a systolic murmur (loudest in the second left intercostal space, and often felt as systolic thrill), dilatation of the right heart (cardiac dulness passing beyond the right edge of the sternum), and hypertrophy of the right side of the heart (often protrusion of the præcordial region). The murmur is not infrequently so loud that it is conveyed beyond the region of cardiac dulness, and may even be heard over the posterior surface of the thorax. It is sometimes heard in the arteries of the neck, with especial frequency and intensity on the left side. It sometimes disappears when the circulation is slow.

If pulmonary insufficiency is present in addition to the stenosis, a diastolic murmur is heard in the second right intercostal space. In pure pulmonary stenosis, on the other hand, the second (diastolic) pulmonary sound is unusually feeble. But if the stenosis is situated at the *conus arteriæ pulmonalis*, the diastolic pulmonary sound, according to Clifford Albutt, is remarkably loud.

*b.* Openings in the auricular septum not infrequently produce no symptoms during life. But the symptoms of a congenital valvular lesion may be so prominent at times that an opening in the auricular septum may be suspected, though it cannot be diagnosed. In a third and rare group of cases, unusual symptoms may arouse a suspicion of such openings. Thus Reisch reported a case of mitral insufficiency in which cervical venous pulse was observed. This was not the result of associated tricuspid insufficiency, but was owing to the fact that the blood which regurgitated from the left ventricle into the left auricle at each systole, passed through an open foramen ovale into the right auricle, and thence into the superior vena cava. Cohnheim reports a case in which thrombosis of a vein of the lower limb was followed by fatal embolism of one of the middle cerebral arteries. A patent foramen ovale had afforded the possibility for dislodged pieces of the thrombus, which had passed along the inferior vena cava into the right auricle, to enter the left auricle, and then the aortic system.

Finally, presystolic murmurs are heard over the heart, loudest at the level of the third and fourth left costal cartilages, although they are conveyed not infrequently to all the cardiac orifices.

It is theoretically conceivable that the symptoms mentioned appear only at

times, when the pressure is abnormally high in the right or left auricle, according as stasis has developed in the domain of the pulmonary artery or the aorta, so that the blood flows through the opening in the septum from the auricle in which the blood pressure is higher into the other.

*c.* With regard to the clinical symptoms of defects in the ventricular septum, only those cases can be taken into consideration in which such defect alone is present. The conditions become more complicated, and cannot be kept apart, when other congenital lesions or inflammatory changes come into play.

Pure cases of the former variety are not frequent. Cases in which the defect is small run their course without symptoms. But symptoms may also be absent when the defects are large. If circulatory disturbances arise, they will consist of the propulsion of a part of the blood of the left ventricle, during systole, into the right ventricle. The physical sequelæ of mitral insufficiency will develop, except that the pulmonary veins and artery are passed by, and the right ventricle is directly affected. Dilatation and hypertrophy of the right ventricle are necessary effects. In addition, a systolic murmur is heard over the right ventricle. But it is evident that these symptoms are too uncertain to permit a positive diagnosis.

*d.* Congenital insufficiency of the tricuspid valves gives rise to a systolic murmur and to venous pulse. A systolic murmur has also been described in stenosis and atresia of the right auriculo-ventricular orifice. This condition, therefore, would be readily mistaken for congenital pulmonary stenosis, if dilatation and hypertrophy of the right heart were not absent, and the murmur were not heard with the greatest intensity at the level of the fourth and fifth costal cartilage, and not in the second left intercostal space.

*e.* The circulatory disturbances caused by patency of the ductus Botalli may be readily constructed theoretically. At each systole blood must flow from the aorta into the pulmonary artery, so that the distribution of the latter is overcharged. This gives rise to increased pressure in the pulmonary artery, dilatation and hypertrophy of the left ventricle, and a clicking quality of the second pulmonary sound. The development of a systolic murmur is also rendered possible. These symptoms will only be present in all their purity if the anomaly exists as an independent affection.

The following symptoms, accordingly, may be looked for: Increased cardiac dulness to the right side; vigorous elevation of the lower half of the sternum; intensification of the second pulmonary sound; a systolic murmur, perhaps thrill, over the heart. The murmur may be conveyed into the arteries of the neck, more markedly in the left carotid than the right. We sometimes find a prominence and elongated area of dulness in the second left intercostal space, corresponding to the dilated and vigorously pulsating pulmonary artery. Abnormally great tension of this vessel may here give rise to an autochthonous systolic murmur and thrill. In aneurismal dilatation of the ductus, disturbances of phonation have been observed and attributed to traction upon the recurrent laryngeal nerve. The symptoms were either present from birth or they developed in later years as the result of certain noxa. We should expect a considerable disproportion between the radial and crural pulse, since the latter loses a part of its aortic supply, and perhaps this sign may be useful in the diagnosis of doubtful cases.



Other congenital heart lesions generally are not susceptible of a local diagnosis during life.

IV. PROGNOSIS.—The prognosis of congenital heart lesions is not favorable. Death usually occurs very early, often immediately after birth. In congenital pulmonary stenosis, it generally occurs before the age of twenty. These conditions are incapable of recovery, and the circulatory disturbances soon assume the upper hand. Some of the patients die with symptoms of phthisis, others succumb to intercurrent diseases, or death is preceded by symptoms of stasis. The fatal termination sometimes takes place during a convulsive seizure.

V. TREATMENT.—This is restricted mainly to dietetic rules. Bodily and mental excitement should be avoided, the food should be nutritious and easily digested, the patient should be hardened by cold rubbings, and “catching cold” avoided by suitable clothing and exercise in the open air. Special conditions must be treated symptomatically, according to well-known rules. Inhalations of oxygen and compressed air have been recommended to relieve the cyanosis.

#### 4. *Thrombosis of the Heart.*

I. ANATOMY.—Clots are found in the cavities of the heart of almost every corpse. Sometimes they are loose cruor clots of a bloody color, sometimes yellowish-gray, elastic, fibrinous clots, occasionally of an osseous appearance. The latter are found most frequently in the right auricle and ventricle, while they are often absent in the cavities of the left heart. But when the death struggle has lasted a long time, they are also found on the left side.

The clots vary considerably in amount. They are particularly abundant in pulmonary affections, especially in fibrinous pneumonia. They sometimes fill the cavities of the right heart to such an extent that the latter appear tense, and the clots not infrequently extend far into the main branches of the large vessels. Their external surface often contains impressions of the semilunar valves or the trabeculae carneae. In many cases, they are entirely destitute of a bloody tinge, in others they appear streaked in places with blood, and a red cruor stripe can be observed with relative frequency upon their lower surface. They evidently do not possess the same characteristics in all diseases. For example, in jaundice they have a distinct icteric color; in leukaemia they are very soft, smeary, thickened, pus-like.

These clots (cardiac polypi) have no clinical significance. They form during the agony or immediately after death when the possibility of the deposition of fibrin is afforded on account of the failing action of the heart.

These clots have nothing in common with the cardiac thrombi which will now be considered. The latter have been called true heart polypi to distinguish them from the pseudo-polypi described above. They can generally be distinguished very readily from one another. The ante-mortem clots are readily separated from the wall of the ventricle, and even where they send prolongations into the meshes between the columnae carneae, they can be readily withdrawn without injury to the endocardium. Cardiac thrombi, on the other hand, are firmly adherent to the endocardium. They cannot be removed without violence, and a loss of substance in the endocardium is left behind. They also vary greatly in appearance and consistence. They form hard, brittle, or

fibrous masses, of a grayish-red, reddish-brown, or pale-gray color. The older they are the more colorless they become, and consequently the superficial layers of large thrombi have the deepest red color. Their surface often looks ribbed, corresponding to various periods of deposition of the fibrin. They are sometimes softened internally, giving rise to the formation of a puriform, chocolate-like, brownish-red fluid, the thrombus thus presenting the appearance of a cyst. These have also been called pus cysts or fibrinous cysts of the heart, though there is no real pus formation in them. On microscopical examination, they are found to contain a few, usually fatty, round cells, which take their origin from the softened thrombus; in addition, there are larger or smaller amounts of hamatoidin, either granular or crystalline. More rarely, calcification of the thrombi develops (heart calculi).

Cardiac thrombi possess certain sites of predilection in the auricles, particularly the auricular appendages, and in the ventricles near the apex. There is a greater tendency to their formation on the right side than on the left. They vary greatly in number; in one case Lebert counted forty in a single cavity of the heart. Thrombi which undergo central softening are apt to be especially numerous. Their size varies from that of the head of a pin or a pea to that of a hen's egg. Small thrombi are sometimes concealed among the columnæ carneæ, so that they are discovered only upon horizontal section of the heart. The thrombi sometimes extend from their site of origin, not alone into the lumen of the adjacent cavity, but also pass through the adjacent orifice, and grow into the next cavity.

Rindfleisch reports a case in which a thrombus, starting from the left auricular appendage, grew through the stenosed mitral orifice, where it presented a constriction, and became swollen into a thick body in the left ventricle. Nobiling reports a case in which the cardiac thrombus passed through the entire aorta into the iliac arteries, and even into the lateral branches of the aorta.

The shape of the thrombi varies. Some are pedunculated, others form round or nodular tumors, which spread with branching roots into the meshes of the columnæ carneæ. The latter are most frequent near the apex of the heart. The rarest form is that in which they form a sort of carpet-like and pseudo-membranous layer upon the inner surface of the heart.

In addition to the cardiac thrombi, the body often presents embolic changes, either in the pulmonary or aortic system, according to the situation of the thrombus in the heart.

II. ETIOLOGY.—The endothelium cells of the endocardium play a prominent part in the formation of cardiac thrombi. So long as they are intact, thrombosis is impossible, but this may occur as soon as the cells have undergone fatty degeneration and desquamation. Thrombi are found in many heart diseases because the endocardium, as a rule, takes part in these changes. The endothelium may also undergo fatty degeneration and desquamation in protracted and exhausting diseases. In addition, certain other conditions may favor the deposition of a thrombus, especially retardation of the current of blood. Thrombosis develops most frequently, therefore, in those places in which stagnation of the blood is most apt to occur, *i. e.*, the auricular appendages and interstices of the columnæ carneæ. Possibly the retardation of the current also impairs the nutrition of the endothelium, so that if the former

is primary, the latter develops secondarily, and thus renders possible the formation of a thrombus.

Some authors believe that morbid increase of fibrin in the blood (hyperinosis), and increased tendency of the unchanged blood to deposit fibrin (inopexia), favor the occurrence of thrombosis, but we have little positive knowledge concerning these two conditions.

Thrombi in the heart are observed under the following conditions:

1. In many cardiac diseases: pericarditis, fatty heart, myocarditis, cardiac aneurism, valvular aneurism, endocarditis. In the latter disease, the thrombi cover the vegetations. The formation of thrombi occurs so much more readily the slower the current of blood, so that the administration of digitalis may sometimes be the cause of thrombosis.

2. In febrile and apyrexial diseases which are associated with great nutritive losses (marantic thrombosis).

3. Bamberger states that pieces of venous thrombi which have been dislodged and carried into the right heart, may remain there and be converted into thrombi. Even in this case we must assume a change in the endothelium of the endocardium by a mechanical or chemical irritant.

III. SYMPTOMS.—In many cases cardiac thrombosis remains altogether latent. In other cases it is concealed by signs of heart failure. We then find: feeble apex beat and heart sounds, irregular action of the heart, syncopal attacks, dizziness, spots before the eyes, ringing in the ears, coldness of the limbs, formication, pallor and livor of the skin, dyspnoea without demonstrable changes in the lungs, etc. In a third series of cases, emboli are formed and enter the arteries of the lungs (hemorrhagic infarctions), the extremities, kidneys, spleen, brain, etc. In still other cases symptoms of a valvular lesion arise. This occurs when the thrombus extends into one of the cardiac orifices and causes stenosis, or interferes mechanically with the unfolding of the valve. Finally, sudden death may occur, if an orifice is suddenly occluded by a piece of the thrombus, or if the latter grows to such an extent as to obstruct the orifice.

The duration of the disease is sometimes only a few days, in other cases a number of months.

IV. DIAGNOSIS.—The positive recognition of the disease is hardly ever possible; the diagnosis can only be made with a certain degree of probability. This is possible if we have an opportunity of making daily, careful examinations; we must rely upon the etiology and the occurrence of repeated embolism and sudden stenosis of the auriculo-ventricular orifice. Gerhardt attaches great importance to a systolic murmur and thrill over the pulmonary artery. These are said to be caused by the pressure of the left auricular appendage, which is filled with the thrombus. But as all these symptoms are rarely present at the same time, it is evident that a probable diagnosis can rarely be made.

V. PROGNOSIS.—The prognosis is unfavorable, as we are unable to effect recovery. Moreover, the thrombi have a tendency to enlarge, and embolism or obstruction of an orifice will result.

VI. TREATMENT.—The question of treatment can hardly be considered, as the disease is not susceptible of a positive diagnosis. We must confine ourselves to the administration of stimulants in conditions of heart failure, and the symptomatic treatment of the other conditions.



### 5. *Tumors of the Endocardium.*

Miliary tubercles, myxoma, gumma, sarcoma, and enchondroma of the endocardium have been described. These conditions are hardly ever susceptible of diagnosis, since they either remain latent on account of their small size or situation, or they proliferate towards the orifices and valves, and give rise to valvular insufficiency and stenosis (the real cause generally remains unrecognized during life), or parts of the tumor break off and give rise to embolic changes.

### *Appendix.*

*a.* Degenerative changes in the endocardium: fatty degeneration, calcification, mucoid degeneration have no clinical significance.

*b.* This is also true of atrophic processes. They give rise to fenestration of the semilunar valve between their free edges and the lines of closure, so that no functional disturbance is produced.

*c.* Congenital anomalies of the valves include supernumerary valves, an insufficient number and inhibited development of the valves. Supernumerary leaflets are not infrequent. They are found more often at the semilunar than the auriculo-ventricular valves, and, according to Crueilhier, are more frequent at the pulmonary than at the aortic valve. As many as five leaflets have been observed at the pulmonary orifice. The leaflets are sometimes malformed, one or the other being merely indicated by a small prominence. Furthermore, they are situated not infrequently at different levels, and may then result in insufficiency. Other congenital lesions of the heart may be associated with these changes, but they cannot be diagnosed during life.

The number of valves is sometimes abnormally small. Functional disturbances will then result unless the remaining leaflets are unusually large and close the orifice. Otherwise the valve will be insufficient.

In some cases a valve may be entirely absent. Ebstein reported a case in which there were merely slight indications of the tricupsid valve. The valvula Thebesii and Eustachii may also present congenital abnormalities. Among one hundred cases, Lauenstein found five in which both valves had coalesced into one.

There may also be an abnormal origin or number of papillary muscles and chordæ tendineæ, and this may give rise, at times, to disturbance in the function of the auriculo-ventricular valves.

*d.* Among congenital malpositions of the heart, we will first mention dextrocardie or dexiocardie. The heart is situated in the right, instead of the left half of the chest; the apex is directed towards the right axillary region; the arch of the aorta passes over the right bronchus and the abdominal aorta runs along the right side of the spine. The thoracic and abdominal viscera are also usually transposed. Some cases have been observed in which a part of the viscera retained their normal position, as in Hickman's case, in which the cæcum alone retained its usual position.

This anomaly is easily recognized. The abnormal situation of the apex beat must arouse suspicion, and this is strengthened by the unusual position of cardiac dulness. The heart sounds are heard with greater intensity on the right side than on the left. But we must distinguish between inversion of the heart and its retraction into the right half of the thorax, as the result of retraction of the right lung.

Whether the other viscera have taken part in the transposition is

readily determined, as a rule. If laryngoscopic examination permits a view of the bifurcation of the bronchi, the entrance to the left bronchus will be found larger than that to the right one, if the lungs are transposed. Vocal fremitus is also greater on the left side, and the intensity of the respiratory murmur is also different. Transposition of the liver is recognized by the greater dulness in the left hypochondrium, while Traube's semilunar space is situated on the right side below the area of cardiac dulness. Splenic dulness is found in the right axillary region. If the stomach is distended with carbonic acid, according to Frerichs' method—a half-teaspoonful of tartaric acid is administered, followed by an equal amount of bicarbonate of soda in water—percussion of the stomach will readily show whether the pylorus of the stomach is situated on the right or left side. If the œsophagus is on the right side of the spinal column, the sounds heard during auscultation of the act of deglutition will be louder to the right of the spinous processes. Transposition of the kidneys will be recognized in the dead body, as a rule, by the lower situation of the left kidney. If the male genitals are transposed, the right testicle will hang lower than the left.

In many cases, the heart has an anomalous position, although the patients do not experience the slightest discomfort. In some instances, the individuals are left-handed. In a third group of cases, finally, other cardiac anomalies are present, and either render life impossible, or soon put an end to it.

Sometimes the heart remains in the median line, as it does in the fetus. This may be associated with other congenital cardiac lesions. Congenital torsion of the heart around its long axis may also occur as the result of inflammatory adhesions.

Protrusion of the heart, *ectopia cordis*, is that condition in which the organ is situated outside of the thorax. In *ectopia cordis pectoralis*, the sternum is divided totally or partially in the middle, and the two halves are more or less separated. In the milder cases, the fissure is covered with integument, beneath which the pulsating heart may be observed. Life may be prolonged for years. In graver cases, there is no integument over the fissure, so that the heart is either covered merely by the pericardium, or, if this is absent, lies entirely exposed. These individuals, as a rule, live only a few hours.

*Ectopia cordis ventralis* is the term applied to the abnormal position of the heart in the abdominal cavity, and is associated with a more or less extensive opening in the diaphragm. The heart is sometimes found in the epigastrium between the stomach and abdominal wall, sometimes in the region of the kidneys, sometimes in a depression in the liver. If a congenital abdominal fissure is also present, the heart is sometimes found, with other abdominal viscera, in an umbilical hernia (*eventratio*). Viability is not excluded in all cases. Peacock reports a case in a man æt. 47 years.

In *ectopia cordis cephalica*, the heart is situated in the cervical region, even at the level of the palate, but this occurs only in non-viable monstrosities.

*e.* Congenital anomalies in the shape of the heart have no significance in many cases. Thaden reported a case in which the left ventricle was prolonged into a finger-shaped projection, 5.5 cm. long, which extended to the umbilicus, and could be felt pulsating through the abdominal walls.

## PART IV.

## NEUROSES OF THE HEART.

1. *Nervous Palpitation of the Heart.*

(*Cardiogmus*, s. *Cardiopalmus*, s. *Hyperkinesis cordis*. *Tachycardia*.)

I. SYMPTOMS.—Nervous palpitation of the heart is the term applied to attacks of accelerated and usually more violent action of the heart, which alternate with periods of normal action, and are independent of organic changes. The attacks last from a few minutes to several hours; more rarely, the affection continues uninterruptedly for days. Sometimes the attacks occur at intervals of months or even years; in other cases, the patients suffer daily, almost hourly. Sometimes only a few attacks occur, sometimes the disease lasts weeks, months, years.

The palpitation is preceded not infrequently by peculiar sensations in the region of the heart. The patients have a feeling of indescribable terror; it often appears to them as if the heart beats irregularly and slow, and threatens to stand still, or they think that something in the heart has torn. During the attack also they complain of anxiety, oppression, dyspnoea; their features clearly reveal the internal misery; the forehead is covered with cold perspiration. The attack begins occasionally with a slight syncopal seizure, or with hemicrania, tinnitus aurium, and vertigo.

The diffuse cardiac impulse is found, during the attack, to be abnormally rapid, lifting, and extensive. In addition, the heart's action is not infrequently irregular. The systolic heart sound is often changed. It is often peculiarly clattering (*cliquetis métallique*), and this is attributed to associated vibrations of the thoracic walls. The first sound of the heart can sometimes be heard with the unaided ear at some distance from the chest as a short ticking. It sounds not infrequently like a murmur during the attack, perhaps because the periodicity of the molecular vibrations of the muscular fibres of the heart suffers on account of the excessively accelerated contractions. The second sound is sometimes so feeble that it appears to be absent; this depends on insufficient filling of the aorta and pulmonary artery, for it appears to grow weaker the more rapid the heart's action, and the less the great arteries are filled. The intervals between the heart sounds are sometimes exactly alike, so that their rhythm assumes the character of a rapid but regular hammering.

The carotids usually pulsate very actively. A systolic murmur and thrill are not infrequently heard over them.

If the attacks last a long time, the cervical veins begin to enlarge and undulate.

The radial pulse is usually frequent, hard, and full; more rarely, soft and small; it is very often irregular. It may even beat more than two hundred and fifty times a minute. When the pulse is so rapid, we should only count every five beats. It is advisable to employ auscultation of the heart, instead of palpation of the pulse, since, when the heart's action is very much accelerated, not every systole of the heart drives a perceptible wave of blood into the radial artery (*pulsus intermittens*).



Respiratory disturbances are hardly ever absent during attacks of palpitation. The patients suffer from a feeling of great dyspnoea, the respirations are frequent and irregular, and are interrupted not infrequently by deep, sighing inspirations. The patient generally assumes certain definite positions, since the dyspnoea is usually increased in the recumbent posture, and is relieved by a sitting or elevated recumbent posture. Speech is jerky, and the words may be uttered in a whisper. Disturbances of deglutition have been observed. Distention of the abdomen, and pain in the gastric region are also mentioned.

The face is reddened and usually covered with perspiration; more rarely the face and limbs are cool, clammy, pale, and livid. The bodily temperature may be temporarily elevated.

The patients complain not infrequently of a rush of blood to the head, dizziness, a feeling of faintness, tinnitus aurium, and sometimes of a peculiar throbbing sensation in the head.

The attack sometimes ends quite suddenly, sometimes very gradually, but the patient suffers for a long time from the dread of its recurrence. The attack may end abruptly after vomiting, eructation, and passage of flatus or faeces. v. Dusch mentions a case in which the attack could be rapidly ended by pressure upon a certain part of the abdomen.

Subjective palpitation of the heart is the term applied to those cases in which the patients present the symptoms described above, and also complain of palpitation; but increased vigor of the heart's action cannot be determined objectively. Often, however, such attacks are accompanied by irregularity of the pulse.

II. ETIOLOGY.—Bamberger attempted to classify the causes of this affection according as the pneumogastric, sympathetic, brain, or spinal cord were the starting-points, but our knowledge of cardiac innervation is too incomplete to permit this plan to be carried out. Proebsting comes to the conclusion that the disease is generally the result of paralytic conditions of the inhibitory fibres of the heart (pneumogastric), more rarely of irritative conditions of the excito-motors (sympathetic). From the standpoint of practical experience, we may distinguish two varieties, according as we have to deal, in the main, with nervous or toxic noxa; but transitional forms are not rare.

Nervous disturbances may start from the brain itself, as when palpitation is the result of mental excitement (joy, sorrow, fright, love, etc.).

Medical students suffer not infrequently from palpitation of the heart, the result of the fear that they are the victims of heart disease. Peter Frank mentions that in writing the chapter on "Diseases of the Heart," he was affected with such violent and persistent attacks of palpitation that he imagined, for a long time, that he was suffering from an aortic aneurism.

In other cases the affection is the result of graver nervous disturbances, viz., softening, hemorrhage, tumors, and congestion of the brain and spinal cord. It is also observed not infrequently in pale, nervous individuals, who suffer from hysteria, spinal irritation, or neurasthenia.

Tumors in the cervical region may act as a cause if they compress the pneumogastric or sympathetic nerves.

The disease is also produced by conditions of profound exhaustion of the nervous system. This category includes mental overwork, excesses in venere, particularly masturbation, chlorosis, loss of blood or nutritive fluids; for example, excessive lactation, and convalescence from severe diseases. Palpitation sometimes occurs in individuals who are affected

later by pulmonary phthisis. Attacks are not infrequent in factory operatives who work long hours in small, poorly-ventilated rooms.

Reflex attacks are very numerous. Thus it is very frequent in gastric affections, in temporary indigestion, as well as in protracted gastric diseases. Sometimes the symptoms appear only after certain articles of diet, or they follow a heavy meal. The irritation is evidently conveyed, in these cases, by the pneumogastric nerve. Constipation or helminthiasis may also give rise to palpitation of the heart. It also occurs not infrequently in those suffering from hemorrhoids, if the customary hemorrhages remain absent. Furthermore, it is frequent in biliary and renal colic, and in many diseases of the uterus and ovaries. According to Remak, diseases of the teeth may give rise to palpitation, through the agency of the trigeminus and sympathetic.

The toxic forms of palpitation include those which result from the excessive use of coffee, tea, or tobacco, and from poisoning with hemlock and henbane. Perhaps this class also includes the palpitation of gouty patients in whom the accumulation of uric acid in the blood may be regarded as the *causa morbi*.

The disease occurs both in children and adults. In the former, it appears usually during attendance at school, as the result of mental strain, ambition, and fear. The disease may also be owing to very rapid growth, disturbed digestion, or helminthiasis.

The individual attack often occurs spontaneously; indeed, the patient is often awakened by it from deep sleep. In other cases it follows mental or bodily exertion. Still others result from spoiled stomach, constipation, anomalies of menstruation, a slight cold, etc. Some patients have attacks of palpitation as soon as they assume the left lateral position in bed; in others the ingestion of certain articles of food has an injurious effect.

III. DIAGNOSIS.—This is not attended with any special difficulty. It is distinguished from the delirium cordis accompanying valvular lesions by the absence of murmurs which, if present at all in palpitation, only occur during the attack, and are always systolic in character, and by the absence of dilatation and hypertrophy of the heart. In chlorotic and anemic individuals, persistent systolic murmurs and slight dilatation of the right ventricle will be observed, but at the same time a hæmic murmur over the bulbus of the internal jugular is hardly ever absent. Delirium cordis following pericardial affections is also differentiated readily from nervous palpitation.

The differential diagnosis from diseases of the heart muscle is more difficult and the diagnosis sometimes must be left undecided until the signs of heart failure decide in favor of the muscular affection. The etiological conditions may possess no slight importance in doubtful cases.

IV. PROGNOSIS.—This depends mainly on the primary affection. In certain cases the primary affection, and with it the palpitation, may be permanently and readily relieved, but in others relief can be afforded only during the attack, while the predisposition remains unaffected. Under such circumstances, the patients not infrequently grow morose and hypochondriacal.

However great the anxiety experienced by a patient during an attack of palpitation, there is no danger, as a rule, of a fatal termination. This may occur only in old people in whom a cerebral hemorrhage may develop after previous degeneration of the cerebral arteries.

As a rule, the heart suffers no injury. It is only in exceptional cases

that hypertrophy develops, as was shown by Corvisart and by Da Costa, who observed the disease often in soldiers (irritable heart).

V. TREATMENT.—In order to relieve the attack as rapidly as possible, the patient should be placed in a large, well-ventilated, and not too light room. He should assume an elevated dorsal position and remove all constricting articles of clothing.

The local application of cold to the cardiac region, in the form of a not too heavy ice-bag, is very useful. The ingestion of small pieces of ice often furnishes extremely rapid relief. But some individuals do not tolerate cold, and in them such measures must be abandoned.

Certain artifices occasionally are very useful. We have already referred to the case in which v. Dusch shortened the attacks by pressure upon a certain part of the abdomen. Waller observes a similar effect from compression of the pneumogastric and sympathetic in the neck. Koelliker, finally, observed in a woman a surprisingly rapid result by advising her to take deep inspirations and then hold her breath.

Among medicinal agents the surest effect is produced by subcutaneous injections of hydrochlorate of morphine (1.0; Glycerin, aq. destil. āā. 15.0. M. D. S.  $4\frac{1}{2}$  syringe-ful), but this remedy should not be given without deliberation. Many other narcotics have been recommended, viz.: opium, chloral hydrate, ether, inhalations of chloroform, aqua laurocerasi, aqua amygdal. amar., tinct. hyoseyami, tinct. aconit., veratrin., belladonna, strychnine, ergot, etc. Large doses of bromide of potassium have also proven serviceable.

In hysterical, hypochondriacal, and nervous individuals, nervines may be very effective. This includes: tinct. valerian. æther., 20–30 drops t. i. d., tinct. castorei canad., 20–30 drops t. i. d., tinct. asafotida 20–30 drops t. i. d.; auro-natrii chlorati 0.3., ext. dulcamar. 3.0. f. pil. No. 30. D. S. 1 pill t. i. d.; argent. nitric. 0.3; argillæ q. s. ut ft. pil. No. 30. D. S. 1 pill t. i. d.; zinc. cyanat. 0.01. 1 pill t. i. d.; liq. kali arsenic., aq. amygdal. amar., āā 5.0. M. D. S. 10 drops t. i. d. after meals, etc.

The exhibition of an emetic is followed occasionally by brilliant results, particularly when the affection is caused by a spoiled and overloaded stomach. (℞ Apomorphin. muriat., 0.1. : 10. D. S.  $\frac{1}{2}$ –1 syringe-ful subcutaneously. ℞ Tartar. stibiat. 0.03, rad. ipecac. pulv., sacch. alb., āā 0.5. M. f. p., d. t. d. No. 3. S. 1 powder every ten minutes until emesis.)

Lemonade, effervescent mixtures, Selters water, champagne with ice may be drunk during the attack.

To prevent a recurrence of the attack we should carefully consider the etiology. Great importance must be attached to a rational regimen: rational exercise in the open air, cold frictions, easily digested and nutritious food, avoidance of all excesses in eating and drinking. If there are no more urgent indications, the prolonged use of digitalis may be very advantageous. (℞ Digital. pulv. 2.0., Ferri lactis 10.0., pulv. althææ q. s. ut ft. pil., No. 50. D. S., 2 pills t. i. d.). Gerhardts recommends the administration of natrum choleineum (for example, 2.0. fiant cum mucilag. gummi arab. q. s. pil. No. 20. D. S., 1 pill every two hours), particularly when digitalis proves ineffective. Change of residence sometimes acts very favorably, particularly wooded mountainous districts which are not too high, and the seashore.

If the patients complain of anæmic and chlorotic symptoms, iron preparations and quinine may be employed. In those suffering from



hemorrhoids, when the customary hemorrhages have ceased, relief is obtained by the application of 5-10 leeches to the anus. In women whose menstrual discharge is scanty, the flow may be stimulated by stimulating foot baths (50-100 gm. of powdered mustard to a bath at 30° R.), 8-12 dry cups to the thighs or 4-6 leeches to the cervix.

The application of the constant current to the pneumogastric and sympathetic sometimes, it is said, affords relief. Fliess recommends the descending current to the pneumogastric, *i. e.*, the anode at the inner surface of the sterno-mastoid, the cathode below, current of moderate strength, applied daily for 1-2 minutes.

## 2. *Nervous Cardiac Pain. Stenocardia. (Neuralgia of the Heart. Angina Pectoris.)*

I. SYMPTOMS—The disease is characterized by attacks of pain which start in the region of the heart and radiate into various nerve districts. It is associated almost always with disturbances in the movements and power of the heart.

The attacks occur sometimes without any exciting cause. They sometimes arouse the patient from deep sleep and occur with special frequency during the transition from the waking to the sleeping state. In other cases they follow a slight cold, bodily or mental excitement, digestive disturbances, etc.

The duration of the attacks varies from a few minutes to several hours, and sometimes the attacks recur so rapidly that the patients are tortured almost uninterruptedly for a number of days. Sometimes months and years elapse before a repetition of the attacks, while in other cases they recur daily. As a rule, the intervals between the attacks become shorter the longer the disease has lasted, and under such circumstances the attacks often increase in intensity.

In the majority of cases the attack begins suddenly. It is preceded more rarely by vertigo, ringing in the ears, nausea, difficulty in swallowing, a feeling of coldness, formication and discoloration of the integument of the limbs.

The intensity of the pain is indescribable. It is located beneath the lower part of the sternum and the region of the left nipple. It is described as cutting, burning, boring, as if a hot iron were whirled around in the heart, as constricting, etc. At the same time the patients experience a sensation as if death were inevitable.

In many cases the heart's action is tumultuous. The number of contractions is unusually large, the apex beat is abnormally extensive and vigorous, and auscultation discloses *cliquetis métallique* in addition to the systolic ventricular sound. In such cases the radial pulse is generally very rigid, but often the contractions of the heart are not all sufficiently powerful to produce a perceptible beat in the radial artery (*pulsus intermittens*). Retardation of the heart's action is observed more rarely. At the same time it grows very feeble; the heart sounds are feeble, the tension of the radial pulse diminishes so that it is hardly perceptible. In extremely rare cases the action of the heart is unchanged.

The symptoms just described are associated with dyspnoea. The patients breathe irregularly and superficially, they gasp and sigh, and believe that suffocation is impending. Such symptoms are chiefly the reflex result of the pain, as the respiratory organs are unaffected, and the patients, when directed so to do, can respire deeply and regularly.

The features reveal the intense terror and anguish. The face is distorted and pale, and generally covered with perspiration.

Neuralgic symptoms or other disturbances of innervation are almost always present in other nerve districts. The most constant one is the violent pain which begins in the shoulder and extends into the left arm. It is sometimes confined to the inner and posterior aspects of the arm (internal and middle cutaneous nerves). Sometimes it extends to the forearm along the distribution of the ulnar nerve. The pain radiates occasionally into the tips of the fourth and fifth fingers, more rarely into the other fingers which are supplied by the median nerve. The patients also complain, as a rule, of numbness and stiffness of the arm, and occasionally of formication, and these sensations often persist for some time after the pains have subsided. In a few cases the pain in the arm has preceded that in the region of the heart. As a rule, the integument is very hyperæsthetic.

Radiation of the pain into the right arm alone is very rare, but both arms are attacked somewhat more frequently.

The pains often radiate to the back of the neck, the occiput and ears, either exclusively or chiefly on the left side. Pains in the distribution of the trigemini are more rare, but Leroux described a case in which the gums were affected.

Pains are observed not infrequently in the upper thoracic region (anterior thoracic nerves). The region of the nipple is also extremely painful in many cases, both spontaneously and on pressure. Cianciosi describes a case in which swelling of the left mamma developed shortly before the outbreak of cardiac pain. Swelling of the left testicle has also been observed in a few cases.

Finally, the pains may radiate to the spinal column, to the umbilicus and the region of the stomach. Pains have also been described in the spermatic cord and testicles, and in one or both legs.

Spasmodic symptoms are also observed, in many cases, in various nerve districts. In the distribution of the pneumogastric we notice spasm of deglutition, vomiting, and laryngeal disturbances. Constant singultus indicates the implication of the phrenic nerve. Even epileptiform attacks may occur.

In the category of vaso-motor disturbances must be included the not infrequent pallor, livid color, and diminished temperature of the integument of the limbs.

Nothnagel maintains that in some cases the vaso-motor disturbances are primary (angina pectoris vasomotoria). He believes that the spasm in the arteries of the skin increases the blood pressure in the aorta, and thus gives rise to the changes in the heart muscle.

Upon the approach of the attack many patients experience great relief if they breathe fresh air. They rush to the window or into the open air. The erect or standing position also affords relief. They often cling to the nearest object, and press it spasmodically against the chest, or press the hands firmly in the region of the heart, or lean the back against some solid support. During the attack some patients demand perfect quiet about them, and slight darkening of the room not infrequently acts very favorably. Involuntary micturition is sometimes observed during the attack. The urine may be very light and watery, and is passed in very large quantities during and immediately after the attack (urina spastica). If the attack lasts for any length of time, faintness is some-

times observed, and may even lead to complete loss of consciousness. At the same time, respiration may be interrupted, the pulse becomes imperceptible, and, if the heart sounds are very feeble, a condition of trance may develop, and be the source of unpleasant mistakes to the superficial observer.

Sometimes an attack suddenly ceases spontaneously. In other cases it disappears after eructation, vomiting, an evacuation from the bowels, or the passage of abundant flatus. Cases have also been observed in which cough and mucoid expectoration appeared at the end of the seizure.

Some authors mention that the seizures may alternate with other nervous disturbances, such as sciatica, hemicrania, or gastralgia.

As a matter of course, we must not confine ourselves to an examination of the heart. Even older writers reported that enlargement of the liver, particularly of the left lobe, gives rise to stenocardia. In some cases mellituria has been observed. Diseases of the kidneys, uterus, and ovaries have also been regarded as etiological factors.

Many patients feel tolerably well in the intervals between the attacks; others suffer from the symptoms of the primary disease. The disease may extend over many years. Not infrequently a condition of marasmus gradually develops to which the patient succumbs. Death also occurs with symptoms of heart failure. Rupture of the heart or cerebral hemorrhage is observed only exceptionally. Sometimes special injurious causes may lead to a fatal termination. Thus, Dickinson reports a case in which a fatal attack developed during coitus.

II. ETIOLOGY.—There are two varieties of angina pectoris, an essential and a symptomatic form. The latter is the result mainly of changes in the circulatory apparatus, particularly of atheromatous changes in the aorta and coronary arteries. It is also found in aortic aneurism and aortic insufficiency, more rarely in mitral lesions. The disease has also been observed in fatty degeneration of the heart and in obliteration of the pericardium, occasionally in mediastinitis and mediastinal tumors. The essential form includes those cases in which no organic changes can be found in the internal organs. Among seventy-one cases collected by Gauthier, only three belonged to this class. It occurs chiefly in young individuals.

Angina pectoris may follow exposure to cold. Thus, angina pectoris vasomotoria is often observed in winter in cold and damp rooms, and in a rough climate.

In other cases it results from mental excitement. Hysteria, hypochondriasis, epilepsy, and insanity have also been mentioned as causes.

A third series of cases are the result of toxic agents, such as the excessive use of tobacco or alcohol.

Certain constitutional diseases, particularly gout and rheumatism, are said to act as causes. Kunze reports a case which was associated with symptoms of secondary syphilis. Hereditary influences sometimes play a part in the etiology.

Angina pectoris is not a very frequent disease. It is pre-eminently an affection of advanced age, probably because diseases of the aorta, coronary vessels, and heart muscle are especially frequent at that age.

Among 75 cases recently collated by Schuetz,

5 occurred before the 45th year.				
6	"	from the 45th-50th years.		
15	"	"	"	50th-60th "
29	"	"	"	60th-70th "
20	"	"	"	70th-80th "



It is remarkably rare in children. René Blache describes a case in a boy of fourteen, suffering from aortic insufficiency and mitral stenosis. v. Dusch reported one in a boy of eleven years, in whom the autopsy showed obliteration of the pericardium and calcification along the transverse sulcus of the heart.

The disease prevails to a remarkably greater degree in males than in females (78.8 per cent according to Gauthier). This is probably owing to the fact that men are much more liable to be attacked by rheumatism, gout, atheromatous degeneration of the aorta and coronary arteries, and fatty degeneration of the heart. This is also true of the excessive use of alcohol and tobacco. The disease is more frequent, accordingly, among well-to-do people, who enjoy freely the pleasures of life.

The bodily constitution appears to play some part in etiology, as the disease is especially frequent in obese individuals.

Climatic conditions also exert some influence, inasmuch as the disease is relatively frequent in colder climates.

Some old reports mention a sort of epidemic development of the disease. In 1824, Kleefeld described an epidemic in Danzig, and Gelineau reported an epidemic on board of a cutter, after the crew had been exhausted in consequence of constant stormy weather.

III. THE NATURE OF THE DISEASE.—Recent writers are inclined to regard the ganglionic system of the heart as the site of the disease. Special importance has been attached to the cardiac plexus, which is composed of fibres from the pneumogastric and sympathetic, and is situated immediately beneath and behind the arch of the aorta. This explains the frequency of angina pectoris in diseases of the aorta. Its frequent association with changes in the coronary arteries is also explained by the fact that the cardiac plexus sends off small branches which form the coronary plexus beneath the epicardium and in the immediate neighborhood of the coronary vessels.

The frequent radiation of the symptoms to the periphery is attributed to the numerous connections of the plexus with other nerve tracts. The predominance of the pains on the left side is, perhaps, the result of the situation of the heart on the left side, and of the specially close anastomosis with the left brachial plexus.

In the majority of cases there seems to be merely a nutritive disturbance of the cardiac and coronary plexuses. Anatomical lesions have been observed in very few cases. Lancereaux reports a case in which the cardiac plexus was congested; furthermore, its connective tissue contained an abundance of white blood-globules which had partly separated the nerve fibres from one another, and had given rise to a condition of beginning degeneration. In another case, Rokitansky described compression of some branches of the cardiac plexus by bronchial glands, and also compression of the right phrenic and left pneumogastric nerves. Haddon also observed compression of the left phrenic by an enlarged bronchial gland, with beginning degeneration in the nerve fibres. Leroux recently reported adhesion of a bronchial gland to the right pneumogastric, but no changes were found in the nerve fibres. Finally, Putjatın found, in one case, proliferation of the interstitial connective tissue with granular and pigment degeneration of the ganglion cells of the auricular septum.

IV. DIAGNOSIS.—Angina pectoris is readily recognized. The paroxysmal cardiac pain is the principal symptom. We should not be satisfied with the mere diagnosis, but should endeavor to ascertain the cause.

V. PROGNOSIS.—This depends on the nature of the disease. If it is

the result of serious disease of the circulatory apparatus, we can hardly hope for improvement. The prognosis is more favorable when special causes, which may be removed, are demonstrable. This is particularly true of the rarer idiopathic forms. Nor is recovery impossible in hysteria, uterine and ovarian diseases, and hepatic enlargement.

VI. TREATMENT.—In treating the individual attacks the patient should be taken into a large, roomy apartment, the window opened, the room slightly darkened, and kept as quiet as possible. The clothes should be loosened. The patients usually assume a sitting, even a standing position.

Many patients experience great relief from swallowing pieces of ice; an ice-bag may be applied to the cardiac region, but it does not act well in all cases.

Subcutaneous injections of morphine act with surprising rapidity and success in many cases. But this remedy is not devoid of danger, and functional disturbances of the central nervous system are particularly apt to occur if the heart has undergone fatty degeneration. Great caution must also be exercised in the use of inhalations of chloroform, sulphuric or acetic ether. Stokes and Bamberger observed epileptiform attacks and severe collapse after chloroform inhalations. Amyl nitrite appears to be indicated in certain cases. This remedy will only be useful when spasmodic conditions in the sympathetic tracts are indicated by coolness, pallor, or livid color of the skin.

Five drops of amyl nitrite are poured on a handkerchief and inhaled until the face grows red, and the patient feels a rush of blood to the head. This remedy is very explosive, and should not be kept near the fire.

Murrell and Ringer have claimed recently that nitro-glycerin (1% alcoholic solution, 1-2 drops every three or four hours) has a favorable effect in angina pectoris. Matthieu and Hay recommend natrium nitrite (0.3-1.0 : 150, one tablespoonful three or four times a day).

If symptoms of heart failure are present during the attack, they should be treated with stimulants (champagne, rum, ether [10 drops on a lump of sugar every half-hour], tinct. valerian. ether., 25 drops on sugar, castoreum, asafoetida, etc.). Mustard hand and foot baths (50-100 gm. mustard to each bath), mustard draughts on the præcordial region, and dry cups on the anterior wall of the chest may also prove useful. In vaso-motor angina pectoris. Nothnagel recommends brushing the limbs, alcoholic frictions (spirit. camphorat., spir. sinapis, spir. angelicæ co., spir. formicarum and chloroform, warm foot, hand, and full baths).

To prevent the return of the attacks, the patient must live rationally. He must avoid excesses in eating and drinking, secure a daily evacuation from the bowels, exercise daily in the open air without exposing himself to cold, harden himself by means of cold rubbings, and avoid bodily and mental excitement.

Injurious influences, such as the abuse of tobacco and alcohol, must be avoided. Hepatic enlargement is treated with laxative mineral waters (Kissingen, Marienbad, Homburg, Carlsbad, Tarasp, etc.). In the same way, treatment must be directed against other existing diseases (uterine and ovarian affections, etc.).

Electricity may be employed if no special organic affections are demonstrable. Duchenne obtained good results in several cases by vigorous faradization of the nipple and cardiac region. The constant current has

recently been employed to advantage, the application being made to the pneumogastric and cervical sympathetic (anode to the neck and cathode to the cardiac region, or, according to Eulenburg, the anode to the sternum and negative pole to the lower cervical spine).

Some patients experience great relief from a change of residence. Wooded, but not too high mountainous regions merit special recommendation, but the sea-shore also acts favorably in some cases. Setons and fontanelles prove useful under certain circumstances.

### 3. *Basedow's Disease.*

I. SYMPTOMS.—This disease presents three cardinal symptoms: acceleration of the heart's action, enlargement of the thyroid gland, and protrusion of the eyeballs. As a rule, these symptoms follow one another, more rarely they develop at the same time; they undergo remissions and exacerbations, and may disappear altogether.

The disease begins, as a rule, with acceleration of the heart's action, the number of beats varying from 120–150 or 200, and in a few cases they are so rapid that they cannot be counted. The patients are also annoyed by a feeling of palpitation, and generally the cardiac impulses are increased in energy. The impulses of the heart are sometimes so violent that they are perceptible through the clothes.

Dilatation of the heart and also hypertrophy are sometimes demonstrable on percussion. On auscultation, we often hear systolic murmurs, and these are perceptible occasionally as systolic thrill. In rare cases Basedow's disease is associated with valvular lesions, which present the ordinary physical signs. In the majority of cases, a systolic murmur must be regarded as accidental, but the auriculo-ventricular valves sometimes present relative insufficiency if the dilatation is very extensive.

Attacks of more marked acceleration of the heart's action are sometimes noticed. These may be associated with pains in the region of the heart, which radiate peripherally. The attacks are also associated, as a rule, with dyspnoea, and sometimes with spasmodic cough.

A few cases have been reported in which the movements of the heart were diminished in frequency.

Subjective and objective palpitation often exists for months, and even years, before the second symptom, enlargement of the thyroid gland, makes its appearance. As a rule, this is not very extensive; one lobe, usually the right, is often larger than the other. At first the organ has a soft, elastic consistence, later it grows firmer and harder. Calcification only occurs when the gland has been previously diseased. Pulsating and vibrating movements are often felt over the gland, and on auscultation we hear a usually continuous humming, which is intensified at each systole of the heart. The thyroïdal arteries are often sinuous and dilated, and pulsate vigorously; the veins are also dilated and sinuous.

The latest of the cardinal symptoms is the protrusion of the eyeballs. Sometimes it is more marked on one side than the other. Cases of unilateral exophthalmus have been observed, but are extremely rare.

The cardinal symptoms usually develop in the order mentioned above. Cases in which the disease begins with enlargement of the thyroid or exophthalmus are not frequent. In rare cases, furthermore, one or the other symptom may be absent. The development of the thyroïdal enlargement and exophthalmus is associated with the action of the heart,



inasmuch as they grow more marked the more vigorous and rapid are the movements of the heart. The symptoms sometimes develop in a single night, and, in other cases, they diminish with equal rapidity.

As a rule, the patients have a delicate constitution. Pale, blonde, blue-eyed individuals, with slight powers of resistance, have a special predisposition to the disease. The psychical condition is very peculiar. Even Basedow noticed that the patients are often remarkably cheerful despite their unhappy position. Very frequently, also, the disease is found in excitable, hysterical, or hypochondriacal individuals. It is also observed in epileptics, and sometimes maniacal or other psychopathic conditions develop. Disturbances in various nerve districts are often observed in Basedow's disease. In one case Sichel observed tremor and paræsthesia of the left limbs, and it is noteworthy that the exophthalmus was also left sided. Eulenburg mentions a case which was complicated with convulsive tic. Hemicrania, trigeminal neuralgia, and occipital pains have been repeatedly described. Finally, complication with chorea has been often observed in children.

Vigorous pulsation of the carotids is usually perceptible. On palpation, the vessel appears strikingly large, and auscultation almost always reveals a loud systolic murmur. As other large arteries are also found to be markedly dilated, we are at once led to think of disturbed innervation of the vascular walls, and to attribute the murmurs to irregular vibrations of the walls. The patients feel the carotid pulsation, not infrequently, as an annoying beating in the head, and this may be the first symptom of the disease. Marked dilatation and pulsation of the abdominal aorta and crural arteries are often noticeable. A systolic sound is heard in the smaller arteries, for example, the brachial artery. Systolic thrill and murmurs are also present.

In a number of cases Libert noticed hepatic pulsation, which he explains by increased arterial fluxion, *i. e.*, as pulsation of the hepatic artery.

The cervical veins are not infrequently found very large and distended. Friedreich observed true venous pulse in them; undulation of the veins has been seen repeatedly. Bruits de diable are often heard over the bulb of the internal jugular, sometimes only during diastole of the heart.

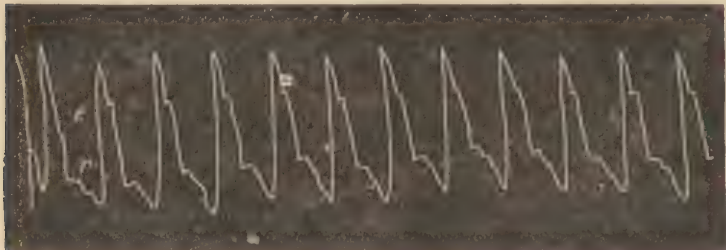
The condition of the radial pulse is not alike in all cases. There is often a very noteworthy difference between the small pulse and the vigor of the cardiac contractions, while in other cases the pulse is sometimes remarkably full and resisting. In the adjacent pulse curve (Fig. 24) the pulse is full and bounding, and shows distinct elevations of recoil, while the elevations of elasticity are not well marked.

Changes in the circulatory apparatus evidently possess intimate relations with certain anomalous secretory processes. In many patients there is a great tendency to profuse perspiration (hyperidrosis), which is occasionally unilateral. Abnormally profuse flow of tears, salivation, and profuse discharge of light-colored urine of low specific gravity are sometimes observed. Eulenburg and Fischer have described glycosuria in this disease. Profuse vomiting or watery diarrhœa is noticed in some patients.

The frequent hemorrhages which have been described are probably the result of disturbances of innervation. These hemorrhages take place from the nose, intestines, lungs, stomach, and occasionally assume a dangerous character.

As a matter of course, all these changes give rise to numerous nutritive disturbances. In one case Friedreich observed a bronze color of the skin; in another, Leube found sclerema of the face and backs of the hands. In the patient represented in Figs. 25 and 26, there was sclerema of the lower limbs and lateral parts of the abdomen; the affected parts

FIG. 24.



Pulse curve of the right radial artery in Basedow's disease. e, elevation of elasticity; r, elevation of recoil.

were light-brown in color. Gangrene of the lower limbs, terminating fatally, has been repeatedly observed.

Among the disturbances of innervation may be included the changes in appetite, for while some patients have an uncontrollable antipathy to food, others have an insatiable appetite (bulimia). Increased thirst is also observed not infrequently.

The bodily temperature is sometimes elevated, indeed the disease may begin with febrile symptoms. The rise of temperature rarely goes beyond 38.5° C. Many patients are tormented by a subjective feeling of heat, which impels them to be constantly near a window. Gluzinski observed that there are sometimes very considerable differences of temperature on symmetrical parts of the body.

The expression of the face is that of astonishment, occasionally of fright. The fixed, peculiar expression is partly the result of the fact that, in consequence of the exophthalmus, the edge of the upper lid does not reach the border of the cornea. In many cases the exophthalmus can be removed temporarily to a slight extent, but this is usually attended with a disagreeable sensation of pain. The exophthalmus is sometimes so marked that the globe is luxated to a certain extent from the orbital cavity, and the eyelid is situated behind the globe.

Græfe called attention to the incongruence between the change in the plane of vision and the associated movement of the upper lid as a very early and extremely important diagnostic sign. It is present even when the protrusion of the globe is not especially marked, and is easily recognized by the fact that the upper lid remains behind on looking downward. Græfe attributes this to the spasmodic contraction of the smooth muscular fibres of the upper lid (muscle of Mueller).

If the globe is rolled downwards forcibly while the upper lid is slightly elevated with the fingers, it is found, in some cases, that little masses of fat project externally under the lid. Dilatation of the blood-vessels and a cyanotic color of the sclera are also mentioned. The mobility of the globe is not infrequently impaired; sometimes there is even marked paralysis of the ocular muscles and double vision. Diminished sensibility of the cornea has been repeatedly described. The condition of the pupils

varies; they may be dilated, contracted, or unchanged. Disturbances of accommodation have been noticed by some writers. Examination of the fundus shows dilatation and sinuosity of the retinal veins. Hyperæmia of the smaller vessels of the retina has also been seen, and Becker sometimes observed pulsation of the retinal arteries.

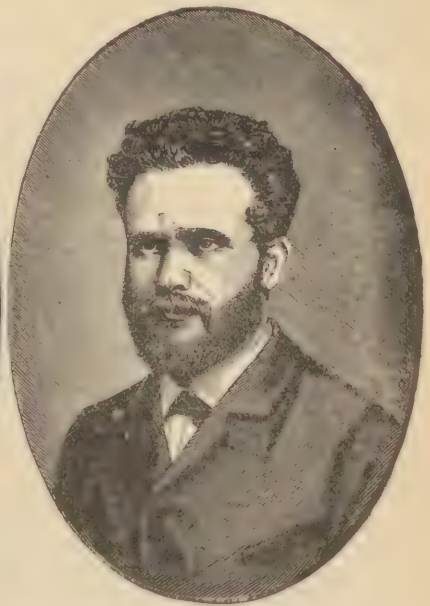
The exophthalmus sometimes gives rise to very serious changes in the eyeball. For since it is the function of the lids, by means of their movements, to cover the globe with tears, and to cleanse it of foreign bodies, dryness, inflammation, and ulceration of the cornea and sclera are apt to develop when the lids are no longer capable of covering the globe. These changes may lead to perforation of the cornea and destruction of the eye. Cornwell maintains that purely trophic changes (attributable to the trigeminus) also occur upon the cornea.

As a rule, the disease begins gradually; its duration is chronic.

FIG. 25.



FIG. 26.



Expression of the face in Basedow's disease.  
Man, æt. 24 years.

The same patient, two months before the beginning of the disease.

Cases with an acute beginning are rare, and it is only in exceptional cases that the disease begins with fever. Still more rarely does the disease run an acute course.

Solbrig described a case in a child, in whom the disease began suddenly, after mental excitement and a sleepless night, but diminished in two days, and had disappeared entirely ten days later.

Exacerbations and remissions of the symptoms occur very commonly. If recovery takes place, the exophthalmus generally subsides last. It is worthy of mention that in a number of cases marriage and childbirth produced recovery.



If the disease lasts a long time, a cachectic condition usually develops, and a fatal termination ensues after increasing prostration. In others, symptoms of stasis develop, and dropsical symptoms precede death. Death may also follow cerebral hemorrhage or intercurrent diseases.

II. ETIOLOGY.—Sex exerts an undoubted influence on the development of this disease. Twice as many women are affected as men. The symptoms of the disease, however, are much more pronounced in men, and, according to Graefe, serious destruction of the eyeball is more frequent in them than in women.

Age is also an important factor. The disease is rare in childhood, so that Dusch was able to collate only eleven cases occurring at this period.

The youngest child was  $2\frac{1}{2}$  years old. The disease develops most frequently from the fifteenth to the thirtieth years. In men, as a rule, it does not begin until a later period.

Heredity seems to have been present in some cases. Solbrig observed the disease in a boy of 8 years, whose mother suffered from Basedow's disease and insanity. Romberg and Juengken observed this affection in two sisters.

The constitution is also an important etiological factor. Delicately built, blonde, blue-eyed individuals, hypochondriacal, hysterical, nervous individuals, chlorotic and anemic persons are particularly liable to suffer from Basedow's disease. Disorders of menstruation are found very often in the female patients, and indeed the symptoms of the disease have been known to disappear as soon as menstruation became regular.

Climatic influences seem to be not entirely destitute of influence. Lebert noticed that the disease was more frequent in Breslau than in Switzerland and France, and Eulenburg observed it more frequently on the Baltic coast than in Berlin.

Exhausting influences, mental excitement, and injuries act as exciting causes.

The disease has been observed after acute infectious diseases, acute exanthemata, intestinal hemorrhage, child-birth, excessive lactation, onanism, and immoderate bodily exercise; also after inanition, intestinal diseases, leucorrhœa, and albuminuria. In other cases the disease has followed joy, sorrow, fright, worry, and injuries to the head.

III. ANATOMICAL CHANGES.—The nature of the disease cannot be inferred with certainty from the anatomical changes. The heart has often been found hypertrophied and dilated. Fatty degeneration of the heart and inflammatory changes in the endocardium have also been described as accidental appearances.

Aneurismal dilatation and abnormal sinuosity of the arteries and varicose dilatation of the veins have been observed in the thyroid gland. Hyperplastic conditions of the interstitial tissue and of the glandular substance have also been noticed. The entire organ is often infiltrated markedly with serum. Calcification and cyst formations are generally of older date and can hardly be brought into direct connection with Basedow's disease.

It has often been noticed that the exophthalmus diminishes considerably after death. This must be explained on the theory that the symptom is owing in part to distention of the retrobulbar vessels and serous infiltration of the orbital adipose tissue. The latter tissue has also been found to be increased in amount. The ocular muscles are

sometimes in a condition of fatty degeneration. Arterio-sclerotic changes in the ophthalmic artery are probably accidental.

Special attention has been devoted to the examination of the nervous system. In a case reported by Geigel, the central canal of the spinal cord was obliterated, and the neuroglia in the vicinity had undergone proliferation.

There is no doubt that the cervical sympathetic may be entirely unchanged in this disease. Abundant development of fat in the interstitial connective tissue, proliferation of the latter, injection of the vessels, atrophy of the nerve fibres and ganglion cells, and unusual pigmentation of these cells have been described in a number of cases, but not all are beyond criticism. Moreover, similar changes are found in other conditions.

French writers have called attention recently to the frequency of enlargement of the tracheal and bronchial lymphatic glands and consequent compression of the pneumogastric nerve.

IV. NATURE OF THE DISEASE.—It seems most probable to us that the disease is the result of paralytic conditions of the sympathetic nerve. The acceleration of the heart's action may be explained by dilatation of the coronary arteries and increased fluxion to the heart. Dilatation of the vessels of the thyroid gland and orbit gives rise to the glandular enlargement and exophthalmus. This explanation does not exclude the assumption that ocular symptoms may develop which must be attributed to irritation of sympathetic fibres. We refer to the contraction of the orbicular smooth muscular fibres which inhibit the movements of the upper lid and, moreover, the development of exophthalmus has been attributed to spasmodic contraction of Mueller's orbital muscle. Dilatation of the pupil cannot be regarded as the result of irritation of the sympathetic, since this symptom is by no means constant and, in addition, it has not been shown to be directly connected with the disease. These difficulties are avoided if we assume special causes for the irritation of the orbital sympathetic fibres. These are found in the exophthalmus. In our opinion this is primarily vaso-motor or paralytic, but on account of the protrusion of the eyeball and the stretching of sympathetic fibres, those nerve tracts which supply the smooth muscles of the orbits are locally irritated.

Some authors believe that irritative and paralytic conditions of the sympathetic are present from the beginning. Geigel, who developed this theory, assumes that the site of the disease is in the spinal centres of the sympathetic. He believes that the oculo-pupillary centre of the cervical cord is in a condition of irritation, while the vaso-motor centre is paralyzed.

Benedict locates the disease in the medulla oblongata. Filehne produced the symptoms of Basedow's disease in rabbits by irritating the restiform bodies.

V. DIAGNOSIS.—The diagnosis is easy if all three symptoms are present. The ocular symptoms are important. We refer to the want of harmony between the movements of the upper lid and the plane of vision, and to the spontaneous pulse in the retinal arteries.

These symptoms also decide the diagnosis when, independently of Basedow's disease, goitre and exophthalmus happen to be associated with one another, the latter symptom perhaps as the result of pressure of the thyroid gland on the cervical sympathetic.

In anæmic and chlorotic individuals, attacks of palpitation of the

heart, associated with enlargement of the thyroid gland, occur very frequently. But the acceleration of the heart's action in such cases is not permanent; the palpitation is the result of bodily or mental excitement.

It must be remembered, moreover, that the movements of the heart may be slowed in certain cases of Basedow's disease.

VI. PROGNOSIS.—The prognosis as regards permanent recovery is not very favorable. The symptoms occasionally disappear for years and then suddenly reappear. As a rule, the prognosis is more grave in men than in women, not alone with regard to the general, but also the local symptoms. Matrimony may be recommended to female patients because, as we have previously remarked, a number of cases are known in which the symptoms disappeared after the first pregnancy.

VII. TREATMENT.—The principal measures in the general treatment of the disease are the prolonged administration of iodide of iron and galvanization of the sympathetic. These measures produced remarkably rapid and permanent effects in the case of the woman whose pulse curve is shown in Fig. 24.

We may prescribe: Ferri iodat. saccharat., 5.0, pulv. et succ. liq., q. s. ut f. pil. No. 50. D. S. One pill every two hours; or Ferri iodat. sacch., 0.1, sacch. alb., 0.5. M. f. p., d. t. d. No. x. One pill every two hours; syr. ferri iodat., syr. simp., āā 25.0. M. D. S. One tablespoonful t. i. d.; Ferri lactici, 10.0, kali iodat., 5.0; pulv. et succ. liq., q. s. ut f. pil. No. 50. D. S. One pill t. i. d. after meals.

In galvanization feeble currents (5–10 elements) should be employed for two minutes every day. The ascending current is applied to the upper part of the cervical cord, the cathode high up in the neck, the anode between the scapulæ. In addition, the anode is allowed to remain on the cervical spine and the cathode applied for two minutes to each cervical sympathetic and pneumogastric. The cathode is first placed high up in the auriculo-maxillary fossa and then gradually drawn along the inner side of the sterno-mastoid towards the clavicle. The medulla oblongata has also been galvanized by transverse currents through the mastoid processes.

Among other remedies we will mention the preparations of iron, quinine, arsenic, bromide of potassium, belladonna, ergot, and the nervines in general.

Great benefit may also be derived from a trip to the mountains or seashore, the use of ferruginous mineral waters, grape and whey cures, sometimes from the cold-water cure. Desnos employed duboisin 0.0005–0.001 daily subcutaneously and reports at least temporary improvement, particularly of the thyroid enlargement.

In women suffering from menstrual disturbances we may attempt to regulate the menses by irritating foot-baths, cups to the thighs, etc., (vide page 143).

Special injurious influences—excessive lactation, bodily and mental over-exertion—must be removed. Meigs attaches great importance to prolonged rest in bed.

Benard observed rapid improvement in three cases after removal of the thyroid gland, and after diminution of the size of the gland by means of cauterization and the passage of a seton. He recommends this plan particularly when the disease begins with thyroid enlargement.

Among the local symptoms palpitation of the heart may require special treatment. We may apply ice bags to the cardiac region and also



give digitalis (2 : 200, one tablespoonful every two hours), but too much should not be expected from the latter remedy.

Bogojowlenki observed good effects from tincture of convallaria (20 drops t. i. d.), and Gerhardt recommends natrium choleinicum (vide page 142).

The thyroid enlargement is often treated with preparations of iodine (sol. kali jodati 10 : 200 one tablespoonful t. i. d.; or tinct. iodin., 1.0, aq. destil., 200, one tablespoonful t. i. d.). These preparations must be used very carefully, as they have been known to produce severe iodism. The galvanic current has also been passed transversely through the tumor or the cathode alone applied to it.

For the treatment of the exophthalmus Graefe recommended the application to the upper lid of iodine (tinct. iodin., tinct. gallarum, aa 5.0. M. D. S.) and iodine ointment (unguent. kali jodat., 5.0. D. S.). If the exophthalmus is so marked that the lids are not closed during sleep, the surface of the eye should be kept clean and moistened four times a day with warm water, lukewarm milk, or diluted glycerin, and a bandage applied at times. Graefe recommended tarsoraphy in very severe cases.

#### 4. *Intermittence of the Heart.*

This is a condition in which the action of the heart ceases entirely for several seconds. It is distinguished from simple irregularity by the fact that when the contractions of the heart again begin, they are regular from the start. Some individuals possess the power of producing intermittence of the heart voluntarily. Donders discovered the solution of this puzzle. By voluntary contraction of the muscles of the neck supplied by the spinal accessory nerve, the fibres of the spinal accessory which enter the pneumogastric are irritated and produce temporary cessation of the heart's action.

Intermittence of the heart has been observed in diseases of the heart muscle, particularly fatty degeneration, in hypertrophy of the left ventricle from aortic stenosis and Bright's disease, as the result of excessive bodily and mental exertion, and in affections of the central nervous system.

The disease sometimes occurs as a sort of independent neurosis, as in the case observed by Skoda and Rokitsansky. The patient, a man æt. 36 years, had several attacks daily of intermittence of the heart. He was aware of the approach of the attack. During the seizure the features were distorted with fear and anguish. A constricting, painful feeling was felt in the region of the heart, and radiated to the side and back of the neck. The return of the heart's action was accompanied usually by a sigh. At the autopsy, cheesy masses were found in the left half of the cerebellum, and the left pneumogastric, right phrenic, and greater cardiac nerves were compressed by lymphatic glands.

Apart from the primary disease, the treatment consists of the recumbent posture and the administration of stimulants.

*Appendix* — Diseases of the coronary arteries. In an organ like the heart, whose most important function is regular and vigorous uninterrupted activity, it is unnecessary to say that serious impairment of the action of the heart will occur as soon as its supply of blood is diminished by narrowing or occlusion of its nutrient vessels. Experimental investigations have shown that after compression, ligature, or artificial embolism of the coronary arteries, the movements of the heart become irregu-

lar and soon cease. Furthermore, cases of sudden death have been reported in which the autopsy revealed nothing beyond stenosis of the coronary arteries, generally as the result of atheroma, more rarely of embolism. Such cases must not be mistaken for those in which sudden death follows hæmopericardium from the rupture of an aneurism of the coronary artery.

In other cases, stenosis of the coronary arteries is characterized by conditions of cardiac insufficiency, sometimes with an acute, sometimes with a subacute course. Asthmatic symptoms, anginal attacks, symptoms of stasis or chronic myocarditis are remarkably frequent in such cases. The proper recognition of these cases is very difficult because similar conditions may arise from other causes. The very intimate relations between diseases of the coronary arteries and those of the muscular tissue of the heart are readily understood from an anatomical standpoint, for the former very often give rise to changes in the heart muscle.

Sclerosis and atheroma are often observed in the coronary arteries, likewise inflammatory thickening of the intima (endarteritis obliterans)—conditions which often give rise to the formation of thrombi. The phenomena of anæmic necrosis appear in that part of the heart muscle whose blood-supply is cut off by the thrombus. If the thrombus forms quite suddenly, the signs of hemorrhagic infarctions may be combined with the necrotic process. Softening of the heart muscle (myomalacia) is sometimes the cause of sudden death under such circumstances because the softened spot is perforated and the heart ruptured. In other cases there is gradual absorption of the softened spot, which is converted into a myocarditic cicatrix. The heart often becomes hypertrophic and dilated, and a chronic aneurism of the heart may form. Not infrequently a valvular lesion develops as the result of sclerosis and atheroma of the tunica intima of the aorta and the endocardium, also cirrhosis of the kidneys from sclerotic changes in the renal arteries.

As a rule, the disturbances in question occur beyond the age of fifty, and more often in males than in females. At times heredity appears to exert an influence. Other cases are the result of alcoholic excesses, gout, syphilis, or violent psychical excitement. The diagnosis, which can only be made with a certain degree of probability, depends chiefly on the etiology and the demonstration of sclerosis and atheroma of the peripheral arteries. A clicking, tympanitic quality of the second sound over the aortic valves indicates calcification of these valves, and we will be more inclined, accordingly, to attribute other disturbances to sclerosis of the coronary arteries. The prognosis is unfavorable. The treatment is purely symptomatic.

## PART V.

### DISEASES OF THE AORTA.

#### 1. *Aneurism of the Aorta.*

I. ANATOMICAL CHANGES.—Aneurism is the term applied to circumscribed dilatations of the vessel. According to the external appearance, aneurisms are divided into sacculated, cylindrical, and spindle-shaped varieties. Their size varies from that of a pea to that of a man's head. The walls of the sacculated aneurism are not always even; on the con-

rary, they are usually nodular and covered with numerous secondary and tertiary dilatations.

The aneurisms may be axial or peripheral. In the axial form, the entire circumference of the vessel takes part in the dilatation; in the peripheral form the dilatation involves only a circumscribed, lateral portion of the wall. The latter variety is not infrequently constricted directly below the opening into the aneurism. If this neck of the aneurism is sufficiently long and yielding, the aneurismal dilatation may be deflected upwards or downwards, and give rise to compression and stenosis of the vessel itself or adjacent organs.

An aneurism is hardly ever found empty. It usually contains more or less firm thrombotic deposits. These are stratified; the oldest strata, which are recognized by their great firmness and gray or grayish-yellow color, are situated next to the inner surface, while the most recent ones are directed towards the free space. The microscope shows that the thrombi contain scattered white blood-globules, more or less changed red globules, and frequently crystals of blood pigment in a fibrillary basement substance.

The thrombi sometimes undergo secondary changes. They are occasionally calcified or softened, the softened masses constituting a pus-like or chocolate-brown fluid. Not very infrequently the thrombi are penetrated by canalicular passages, which are supplied with blood from the aneurism itself.

The thrombi are more abundant in sacculated than in cylindrical and spindle-shaped aneurisms. This is owing to the fact that the circulation is especially slow in the sacculated aneurism. The thrombotic deposit is induced by the changes in the tunica intima which are rarely absent in an aneurism.

The formation of thrombi within an aneurism is not an unfavorable process, inasmuch as it thickens the wall, and thus tends to prevent rupture. Indeed, in smaller aneurisms, the sac may be entirely occluded, and spontaneous recovery ensues. On the other hand, the thrombi are a source of danger, inasmuch as parts may be separated from them, enter the circulation, and be carried to the periphery as emboli. Thrombi may also occlude the entrance to vessels which enter the aneurism. The aneurism itself may also give rise to distortion and slit-shaped distention of the mouths of the vessels.

The aneurism is usually the result of dilatation of all three coats of the vessel (true aneurism). It generally begins with endarteritic changes and fatty degeneration, the muscular coat diminishes in extent, and also in tonus and power of resistance, and thus allows partial dilatation of the trunk of the vessel. But calcification and fatty degeneration of the tunica intima of an aneurism are no proof that the development of the aneurism was preceded by arterio-sclerotic changes, since they may have developed secondarily. Moreover, as arterio-sclerosis is very common and aneurisms are rare, the etiological significance of endarteritic processes seems to have been much exaggerated.

Koester has recently maintained that aneurisms are the result of mesarteritis, *i. e.*, primary inflammatory disease of the muscular coat. He finds that the chronic inflammatory process is confined to the nutrient vessels which pass from the adventitia into the muscular coat, and that they become surrounded with fibrous bands which cause destruction



of the muscular tissue. It is evident that these changes must favor the dilatation of the vessel.

On microscopical examination, the muscular coat in the wall of the aneurism is found extremely thin or altogether absent. The adventitia and intima may be unchanged, or they are rich in cells and thickened. If the aneurism continues to grow, the intima grows thin and finally disappears, and then similar changes appear in the adventitia. Under these conditions rupture of the aneurism must occur, or adjacent organs and tissues must supply the deficiency in the wall of the vessel.

An aneurism is dangerous on account of its tendency to constant enlargement. It is evident that it must soon reach a point at which rupture takes place or the integrity of adjacent organs is disturbed by compression. The lungs are very often compressed by intrathoracic aneurisms. Cases have been reported in which, after rupture of an aneurism, the blood first entered the lungs, or the lungs with the greatly thickened pleura had coalesced with the lumen of the aneurism. The main bronchi, particularly the left one, and the trachea very often undergo compression. Not infrequently the pneumogastric and recurrent laryngeal nerves are compressed, and undergo degenerative atrophy. The auricles and venæ cavæ may also undergo compression. The same fate may also befall the osseous tissues. Erosions develop at the manubrium, clavicles and ribs, and may lead to entire disappearance of portions of these bones.

The joints are often destroyed during this process, for example, the sternoclavicular articulation. The microscope shows that the eroded portions of the bone, after having lost their lime salts, assume a peculiar fibrous structure.

If the aneurism perforates the bones, it is first covered by the muscles of the thorax. But these gradually disappear, so that the aneurism is situated immediately beneath the integument. Rupture then occurs in a short time, either suddenly as the result of bodily exertion or from gradual thinning or gangrene of the skin. The rupture may also take place into the pericardial or pleural cavities, the lungs, bronchi, trachea, venæ cavæ, pulmonary artery, and cavities of the heart.

The phenomena of compression are still more complicated when the aneurism forms in the abdominal cavity. The stomach, intestines, liver, and kidneys are then involved. Rupture may take place into the stomach and intestines or into the urinary passages. Morbid changes develop not infrequently in the spine and spinal cord. Gradual erosion of the spine occurs, so that the aneurism is in contact with the spinal meninges. If rupture then takes place, the blood passes upwards and downwards in the spinal canal, or between the meninges, or it gives rise to destruction of the tissue of the spinal cord itself. In other cases there is simply compression of the cord, resulting in circumscribed softening and secondary degeneration of certain columns of the cord.

Aneurisms of the thoracic aorta are much more frequent than those of the abdominal aorta.

The following is the proportion among 234 cases collated by Crisp :

Thoracic aorta.....	175=74.8 per cent.
Abdominal aorta.....	59=25.2      “

Crisp found the following relative frequency in the affection of the different parts of the thoracic aorta :

Ascending aorta.....	98=58.8 per cent,
Arch of aorta....	48=28.7   “
Descending aorta.....	21=12.5   “
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According to Lebert and Myers, aneurism of the arch of the aorta is a little more frequent than that of the ascending portion.

Aneurisms of the aorta develop particularly in those localities against which the current of blood impinges with special force. In the ascending aorta and arch, this position is the anterior and convex surface, in the descending and abdominal aorta, the posterior and lateral surfaces.

II. ETIOLOGY.—Age exerts great influence on the development of aneurisms. They are exceptional during the first decade, most frequent from the thirtieth to fiftieth years. This is owing to the fact that diseases of the arteries usually begin at an advanced age.

Congenital aneurisms are exceedingly rare. Phenomenow recently described a case in which the extraction of the child was much impeded by an abdominal aneurism.

Aneurisms are much more frequent in men than in women, and among the laboring classes than in the well-to-do. The influence of these factors arises from their effect on the development of arteritic and endarteritic affections of the aorta.

Geographical conditions also exert an influence which should not be underestimated. Thus, aneurism is a very common affection in England. This has been supposed to be the result of the widespread abuse of alcohol in that country, but this opinion has been disputed.

According to Liddell's statistics, the natives of the United States suffer much less from aneurism than foreigners, as is shown by the following table :

Natives.....	81 cases = 33.5 per cent
Foreigners....	161   “   = 66.5   “
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A few observations seem to indicate that heredity may exert an etiological influence.

Certain constitutional diseases, especially gout and syphilis, have been supposed to bear a causal relation to the development of aneurisms. According to Lebert, aneurisms may also form under the influence of rheumatism.

Injuries are often regarded as the immediate causes of aneurism and, although their influence has been undoubtedly overestimated, positive cases have been reported in which the first signs of the disease appeared directly after an injury. The trauma may be the result of a blow, push, fall, or the lifting of a heavy weight ; but so far as regards true aneurisms, we must assume that injuries will only prove effective if they have been preceded by changes in the vessels.

We have previously hinted that the excessive use of alcohol exerts an influence on the development of aneurisms, for, like the heart, the blood vessels of drunkards very often present serious morbid changes.

The etiological factors mentioned favor the development of aneurisms by giving rise to fatty degeneration of the intima and to endarteritic changes, but, according to Koester, they give rise to mesarteritic changes. It has also been held that aneurisms may follow vaso-motor disturbances without any anatomical changes in the muscular coat.

**III. SYMPTOMS.**—Even aneurisms of considerable size may not present any symptoms. They are either found accidentally on examination or they give rise to sudden death from rupture in individuals who apparently enjoyed excellent health.

In other cases very grave symptoms arise which may vary greatly according to the organs implicated. The appearance of a pulsating tumor is a very valuable diagnostic sign. According to the situation of the aneurism, it appears sometimes along the right border of the sternum in the upper right intercostal spaces, sometimes it projects beneath the manubrium sterni and above it in the jugular fossa, more rarely it is immediately adjacent to the left border of the sternum. Aneurisms of the descending thoracic aorta generally appear on the dorsal surface to the left of the spine, between the latter and the scapula. Aneurisms of the abdominal aorta also often project on the left of the spine, but if the abdominal walls are thin and sunken, the pulsating tumor may be visible anteriorly.

The tumor may grow to the size of a man's head. In a case described by Bamberger, an aneurism of the arch of the aorta projected upwards to such an extent that the chin rested directly on the tumor. If the tumor is not very prominent, oblique illumination may be very useful in examination, because slight projections then become much more distinct. The growth of the tumor may sometimes be followed from day to day.

The skin over the tumor is usually remarkably glossy and destitute of folds. It is thin, and cannot be raised into folds as readily as over adjacent parts. If the aneurism has grown to such an extent as to threaten rupture through the integument, the latter generally assumes an ominous redness, or a gangrenous process develops with the formation of a scab, after the desquamation of which, fatal hemorrhage may occur.

The pulsating movements often appear more distinct on oblique illumination. These movements alone may render the diagnosis almost certain. This is especially true when the tumor appears upon the anterior wall of the thorax. The suspicion of aneurism must be aroused at once as soon as we have to deal with two circumscribed, independent centres of pulsation, one of which would correspond to the apex beat, the higher one to the aneurism.

On palpation, as a rule, the tumor presents a soft and elastic, yielding consistence. Not infrequently it is extremely sensitive to pressure. Palpation should be practised very carefully, as we are liable to loosen some of the thrombotic deposits in the aneurism, and thus give rise to embolism.

The pulsation is felt as a rhythmical, gradually increasing wave. It is not a simple elevation and depression, but an all-sided systolic enlargement of the tumor. If the fingers are placed in a circle upon the pulsating tumor, they are separated from one another at each elevation.

In aneurisms of the ascending aorta and the arch, we often find not a single but a double impulse. Auscultation shows that the second (usually feebler) impulse coincides with the second (diastolic) aortic sound, so that the systolic impulse corresponds to the filling of the aneu-



rism, and the diastolic impulse is conveyed from the aortic semilunar valves.

The force of the systolic impulse may also be important in diagnosis, for if the pulsating tumor shows more vigorous impulses than the apex beat, the former must possess special pulsating powers and processes.

Purring thrill is found not infrequently over the aneurism. It is almost always systolic in character, diastolic thrill (associated with the former) being very rare.

In aneurisms of the arch which have not given rise to a visible prominence, palpation may be very important. When the finger is pressed downwards deep into the jugular fossa, it sometimes reaches the aneurism.

Percussion furnishes very valuable data. In many prominent aneurisms it permits a definition of adjacent organs, thus enabling us to form an opinion of the approximate size of the aneurism. Latent aneurisms are sometimes disclosed by the unusual dullness which is present to the right or left of the sternum, over the manubrium sterni, or along the spinal column. Aneurisms of the abdominal aorta can only be reached from the anterior surface if the abdominal walls are thin and yielding, and the intestines filled, in the main, with gas. Considerable pressure with the pleximeter will be necessary in order to compress the intestines lying over the aneurism, and thus to eliminate their tympanitic percussion note. It may be advisable to evacuate the contents of the bowels, by means of laxatives, before the examination.

Auscultation does not furnish the same symptoms in all cases. We may hear a simple systolic sound, a systolic and diastolic sound, a systolic murmur, or a systolic and diastolic murmur. The murmurs are sometimes so loud that they are audible at a distance from the patient. According to Graves, they are intensified in aneurisms of the abdominal aorta, when the pelvis is kept as high, and the thorax as low as possible.

From theoretical considerations, it would seem that all aortic aneurisms should give rise to systolic murmurs, because the aneurism constitutes a sudden dilatation of the blood channel, in which whirling motion of the blood and, therefore, a murmur should arise. The murmur should only be absent if the dilatation is very gradual (spindle-shaped aneurisms), or the thrombi on the inner surface of the tumor are so abundant that a sudden dilatation of the channel is, in reality, no longer present, or finally, if, on account of cardiac weakness, the rapidity of the circulation in the aortic system is exceedingly slow. From the last-mentioned possibilities, it is evident that aneurismal murmurs may disappear permanently or temporarily.

If a systolic and diastolic sound are heard over an aneurism, the former is explained by the tension of the aneurismal wall, and the absence of the conditions necessary to the development of systolic murmurs, which were mentioned above. The diastolic sound, on the other hand, must be interpreted as conveyed from the aortic semilunar valves. This view is corroborated by the fact that it is heard so much more frequently the nearer the aneurism is to the origin of the aorta. For this reason, double sounds will hardly ever be heard over aneurisms of the abdominal aorta. This explanation, it is true, seems to be contradicted by the fact that the second sound over the aneurism is not infrequently louder than that over the origin of the aorta, and of different quality; but acoustic modifications in sounds conveyed from the aortic valves are very readily produced by resonance within the aneurism.

The occurrence of systolic and diastolic murmurs over an aneurism does not always depend on the same causes. If, in addition to the aneurism, there is aortic insufficiency, the diastolic murmur which is formed in the left ventricle is sometimes conveyed to the aneurism. We must assume this mode of development if the murmur at the origin of the aorta and that over the aneurism have the same acoustic quality. But if the aortic valves are sufficient, the conditions for the production of the diastolic murmur must be present in the aneurism itself. These conditions consist of the phenomena of regurgitation of the blood. If the blood which is propelled towards the periphery regurgitates into the aneurism during diastole of the heart, whirling movements of the blood and a diastolic murmur must develop in the aneurism if the current of blood is sufficiently rapid.

Severe subjective disturbances are felt not infrequently at the site of the aneurism. The patients complain of annoying throbbing and beating, often associated with severe shooting or burning pains. The symptoms very often are intensified in certain positions of the body, particularly left lateral decubitus. On the whole, pain plays a prominent part among the subjective symptoms of aneurism. It appears as brachial or intercostal neuralgia, or as extremely violent pains in the abdomen. The latter form may simulate gastralgia, hepatic or renal colic. At all events, it must be remembered that protracted and violent neuralgic symptoms should rouse the suspicion of aneurism if other causes may be excluded.

Changes in the heart are observed very frequently. The aortic valves are not infrequently insufficient because the endarteritic process which gave rise to the formation of the aneurism extends to the aortic valves and renders them incapable of function. Mitral lesions are much rarer, though these valves often present endarteritic changes at the autopsy.

The condition of the heart muscle is not alike in all cases. If certain valves are incapable of function, those changes must necessarily develop which are to be expected according to the laws of compensation. These generally consist of hypertrophy and dilatation of the left ventricle. These changes in the left ventricle will also develop if extensive arteriosclerosis has long preceded the development of the aneurism. But if the conditions are uncomplicated and the aneurism alone is present, changes in the heart muscle may be entirely absent. Indeed, Axel Key has shown that the muscular tissue of the left ventricle then undergoes dilatation and atrophy. This fact evidently is not in accord with the theoretical considerations according to which, since the sudden dilatation of the blood channel within the aneurism offers greater resistance to the left ventricle, hypertrophy or dilatation of the left ventricle should be expected. Axel Key explains this on the ground that the expected hypertrophy remains absent on account of the anæmia and imperfect formation of blood, and in addition, an aneurism which is situated near the heart generally presses on the adjacent pulmonary artery. The left ventricle therefore receives very little blood from the pulmonary veins, a circumstance which antagonizes the conditions necessary to hypertrophy. Hypertrophy of the right ventricle is not infrequent. The origin of the pulmonary artery is almost always found to be dilated. Inflammation of the pericardium occurs not infrequently during the course of aneurisms.

The heart is very often found to be dislocated. In aneurism of the ascending aorta and arch, the heart may be pushed downwards and to the

left externally, so that the apex beat is in the sixth intercostal space outside of the line of the left nipple. In aneurism of the descending aorta the heart is not infrequently pushed upwards and internally, and the dislocation is more marked the nearer the aneurism is to the diaphragm and the greater its dimensions.

The heart's action is generally irregular. Attacks of palpitation occur, associated with dyspnoea, and occasionally with violent pains in the arms, back of the neck, and epigastrium. These seizures are entirely similar to angina pectoris. They occur sometimes in certain positions of the body, and many patients are comfortable only when they assume an elevated dorsal or a sitting posture.

If the heart's action is very excited, throbbing of the carotids becomes noticeable. According to Franck, the radial pulse very often presents the characteristics of *pulsus paradoxus* s. *inspirations intermit-tens*, *i. e.*, the pulse becomes considerably smaller or imperceptible during deep inspiration. This is said to occur only in aneurisms of the arch, and to affect the pulse of those vessels alone which take their origin from the aneurism. Franck explains this phenomenon on the theory that the aneurism presents an unusually large surface to the influence of the intrathoracic pressure, and he believes that the limitation of the paradox pulse to certain vessels may be employed in the diagnosis of the situation of the aneurism.

Great significance attaches to the retardation of the pulse as compared to the apex beat, and to retardation and unequal character of the pulse in symmetrical arteries of the body. If the aneurism is situated on the ascending aorta, all the pulses of the periphery of the body will be delayed in comparison with the apex beat. In aneurism of the arch and descending aorta, the pulse in the neck and arms will coincide with the apex beat, the crural pulse will be considerably delayed. In aneurisms, particularly of the arch of the aorta, there are often differences in fulness and in the time of occurrence of the pulse between corresponding arteries on both sides of the body. These are often caused by the distention and slit-shaped narrowing of a vessel at its entrance into the aneurism. But even when the mouths of the vessels are unchanged, the aneurism itself furnishes the condition for the production of these changes. If an aneurism is situated between the origin of the innominate and left carotid arteries, the pulse in the right carotid and radial arteries must evidently appear earlier than that in the arteries of the left side of the neck and head, the left arm, and both lower limbs. If the aneurism is situated between the left carotid and subclavian arteries, the apex beat, carotid, and right radial pulse will coincide in point of time, while the pulse in the left radial and both crural arteries will be delayed. Whether the change in the pulse is the result of stenosis of the mouth of the vessel or of the situation of the aneurism can be determined in many cases from the fulness of the pulse. The latter will be considerably diminished if the mouth of the vessel is narrowed.

Ophthalmoscopic examination shows spontaneous arterial pulse in the retina, sometimes more distinctly or exclusively in one eye. Lebert and Quinke also observed capillary pulse in the face, *i. e.*, the face was reddened with each cardiac systole. According to Quinke, this phenomenon is noticed only in large aneurisms.

The cervical veins not infrequently present marked distention and undulatory movements. If the superior vena cava is strongly compressed



by an aortic aneurism, the subcutaneous veins of the neck and upper part of the thorax, which serve as collateral tracts, are often very sinuous and dilated. If an innominate vein alone is compressed, the enlargement of the cutaneous veins will be confined to one side. Finally, if the pressure affects the inferior vena cava, the veins of the abdominal walls will be dilated.

The dilatation of the veins is often followed by œdema, its situation varying according to that of the dilated veins. Compression of the inferior vena cava may be followed by albuminuria and signs of passive congestion of the kidneys.

Numerous disorders of the respiratory tract appear during the course of aortic aneurism. Very many patients complain of dyspnœa, the result of compression of lungs. The dyspnœa is so much more dangerous the larger the aneurism, the more rapid its growth, and the more unyielding the thorax. The dyspnœa sometimes follows compression of a main bronchus. The left is compressed more frequently and is sometimes entirely occluded. The respiratory movements on the side involved are remarkably diminished in extent, and this side of the thorax may present inspiratory retraction of the intercostal spaces. Vocal fremitus is diminished or extinguished. On account of the diminished tension of the lungs on the side in question, a deep tympanitic note is heard on percussion, and dulness develops if the lung collapses entirely. On auscultation the respiratory murmur is found to be feeble or altogether absent; in the former event sibilant and sonorous râles may be heard.

Very important changes may be recognized on laryngoscopic examination. One or both vocal cords are not infrequently found to be immovable, as the result of unilateral or bilateral paralysis of the recurrent laryngeal nerve. If the paralysis is unilateral, the left nerve is generally affected: if both are paralyzed, the voice becomes aphonic, the patients are unable to cough vigorously, and often swallow "the wrong way," because closure of the entrance to the larynx is impaired by paralysis of the epiglottis.

If the aneurism presses against the trachea, vigorous pulsating movements may be shown by the laryngoscope, at the site of compression. It must be remembered, however, that slight pulsating impulses of the tracheal wall have been described as normal. If these impulses are very vigorous, the patients may be tormented by a feeling as if the laryngeal tissues were dragged rhythmically downwards.

Asthma-like attacks develop occasionally, and are attributed to temporary irritation of the pneumogastric. They may be associated with vomiting and stenocardic symptoms.

Pulmonary phthisis develops not infrequently. Among 77 cases of aneurism of the arch of the aorta collected by Hanot, the condition of the lungs was not referred to in 35; but among the remaining 42 cases phthisis occurred 18 times (38.1 per cent). Some writers attribute this complication to compression of branches of the pneumogastric, others to compression of the pulmonary artery.

Very distressing symptoms on the part of the nervous system very often make their appearance. The most frequent one is brachial neuralgia, especially in the left arm. It is the direct result of the pressure of the aneurism on the brachial plexus, and may be associated with numbness, formication, weakness, and even paralysis of the arm. Intercostal neuralgia is not infrequent in aneurism of the descending thoracic aorta. Aneurism of the abdominal aorta gives rise to violent pains in the spine.

If the spinal column has been eroded, convulsions, paræsthesiæ, and paralyzes of the lower limbs appear, and sometimes paralysis of the bladder and rectum.

Not every functional disorder of the upper limbs must be attributed to paretic or paralytic conditions. The mobility of the limbs may also be impaired by the destruction of the sterno-clavicular or scapulo-clavicular articulations, or by erosion and widespread destruction of the scapula.

Aneurisms have been found not infrequently in the insane. Manson and L. Mayer state that aneurismal dilatation of the internal carotid is a frequent lesion in the insane. According to Mayer, the psychosis is the result of the circulatory disturbances in the brain produced by the change in the vessel.

Many patients suffer from extreme insomnia. A difference in the pupils is sometimes noticed, and is attributable to irritation or paralysis of sympathetic fibres.

Morbid symptoms on the part of the abdominal organs must be looked for, above all, in aneurisms of the abdominal aorta. Changes in the thoracic viscera are also observed at the same time.

Neuralgic symptoms may simulate gastralgia. Not infrequently there are attacks of violent vomiting, which have been attributed to intermittent irritation of the pneumogastric. Attacks of colic have been described with equal frequency. Pressure upon the intestines may interfere greatly with defecation, and cases have been reported in which the feces had the ribbon-like shape observed in intestinal cancer.

Narrowing of the œsophagus merits special mention. In old people there may be a suspicion of œsophageal cancer; but we should hesitate to introduce the œsophageal sound before we are sure of the absence of an aneurism of the aorta. In the other event, the aneurism may be perforated, and give rise to a rapidly fatal hemorrhage. The disturbances in swallowing sometimes occur in paroxysms, or only in certain positions of the body, for example, in dorsal decubitus.

Obtuse forms of jaundice, the result of pressure on the ductus hepaticus, or ductus choledochus, have been repeatedly observed.

In one case, Ralfe described increased production of urine. Diminished diuresis may also occur if the aneurism compresses the ureter and produces stasis of urine and dilatation of the ureter above the site of compression.

Cases have been reported in which an aneurism was present for ten, twenty years, or even longer. Sometimes, indeed, the patients are able to do heavy work for a long time. From a large number of observations, Lebert estimates the average duration of the disease at fifteen to eighteen months.

Spontaneous or artificial recovery of an aneurism can hardly ever be looked for. The fatal termination occurs in various ways. Sometimes the patients grow very pale and cachectic, hemorrhages appear occasionally in the skin and mucous membranes, œdema develops, and death follows after symptoms of general marasmus. In other patients the stenosis of the œsophagus increases to such an extent that death takes place from inanition. Compression of the trachea, bronchus, or lungs may also cause death by suffocation. Again, intercurrent diseases, especially pneumonia, pleurisy, or pericarditis, are the immediate causes of death. This may also follow grave icterus and spinal paralysis. Em-

holic processes, particularly in the arteries of the brain and limbs, are also of grave significance.

Death is often the result of rupture of the aneurism. Rupture externally is usually preceded by thinning and reddening of the skin, and the formation of a gangrenous scurf. The hemorrhage sometimes ceases spontaneously or is checked by artificial means, particularly if the blood is oozing very slowly. Rupture into the pulmonary artery or one of the cavities of the heart is revealed by the abnormal murmurs and the circulatory disturbances, but such processes may be tolerated for a remarkably long time. Aneurisms of the ascending aorta often burst into the pericardial cavity. The symptoms of internal hemorrhage then arise, the cardiac dulness is rapidly increased, the movements of the heart are paralyzed. In rupture of an aneurism into the pleural cavity, the symptoms of internal hemorrhage are soon followed by those of pleural effusion (dulness, diminished or abolished vocal fremitus, etc.). Rupture into the lungs, a bronchus, or the trachea, is usually followed by fatal hemorrhage from the lungs. Uncontrollable hæmatemesis or profuse intestinal hemorrhage is evidence of perforation of the aneurism into the œsophagus, stomach, or intestinal canal. Rupture into the urinary passages gives rise to hæmaturia.

IV. DIAGNOSIS.—We must consider not alone the diagnosis of the aneurism as such, but also of its situation.

When the aneurism is latent, suspicion of its existence will be roused only by the presence of special symptoms. These include unilateral or bilateral paralysis of the recurrent laryngeal nerve, difficulty of deglutition, obstinate neuralgia. The diagnosis of an aneurism can only be made under such circumstances if other causes for the symptoms mentioned can be excluded. In some cases, murmurs found over a circumscribed part of the aorta may be important in diagnosis. But we must assure ourselves that the murmur is not the result of compression and stenosis of the aorta.

If the aneurism has given rise to the formation of a pulsating tumor, it may be mistaken for a solid tumor situated upon the aorta, which conveys its pulsations to the former. If the movement is simply conveyed, the tumor rises and falls, moves up and down, or from the right to the left, while the pulsating movement of an aneurism is conveyed in all directions. If the aneurism can be grasped between the fingers, its volume is found to increase at each systole, while the dimensions of a tumor situated upon the aorta remain unchanged. Finally, an aneurism presents a gradually increasing systolic increase in volume, while the pulsating movement of a tumor is abrupt and vigorous, and disappears with equal abruptness.

In a case reported by Meyer, an aneurism, which had produced complete occlusion of the left bronchus, was overlooked, and the disease regarded as a left pleurisy. In such cases, special attention should be paid to abnormal murmurs and pulsation, and to differences in the time of occurrence and the strength of the peripheral pulse.

The situation of the aneurism is decided in the majority of cases by the position of the thoracic tumor. Aneurisms of the ascending aorta generally appear at the right edge of the sternum at the level of the second and third intercostal spaces. Those growing from the arch appear generally under the manubrium sterni or along the left edge of the sternum. Aneurisms of the descending thoracic aorta are situated



usually to the left of the spine, most frequently at the level of the eighth dorsal vertebra.

Comparison of the peripheral pulses may also be employed for accurate localization of the aneurisms, particularly of the ascending aorta and arch. The points to be considered in diagnosis are: Delay of the pulse compared with the apex beat, non-synchronous occurrence of symmetrical pulses, dissimilar quality of corresponding pulses, partial "paradox pulse."

The differentiation between aneurism of the arch of the aorta and the innominate artery may be very difficult, and even impossible. Aneurisms of the innominate generally appear under the first right costal cartilage and further up beneath the clavicle, they press towards the right supra-clavicular fossa, and give rise to changes in the pulse of the right side of the neck and head, and of the right arm.

V. PROGNOSIS.—The prognosis of aortic aneurisms is unfavorable. Spontaneous recovery can hardly be hoped for, and the application of artificial measures is neither certain nor destitute of danger. As the aneurism, moreover, has a tendency to enlarge, there is danger of final rupture or compression of vital organs.

VI. TREATMENT.—In the majority of cases, therapeutics must be restricted to rational dietetic measures and the treatment of specially distressing symptoms. The patients must avoid all mental and physical excitement, eat easily digested and nutritious food, and secure a daily evacuation from the bowels, if necessary by the aid of laxatives and mineral waters. Any bodily exertion may be the immediate cause of rupture of the aneurism. Narcotics (subcutaneous injections of morphine) must be employed to combat violent pains and stenocardic and asthmatic attacks. When severe palpitation and signs of heart failure develop, we may prescribe digitalis and the local application of cold to the heart.

Ferruginous preparations and quinine are indicated in anæmia. If the aneurism projects prominently externally, it should be guarded against pressure and injury by a suitable bandage. When rupture impended, good effects have been seen from applications of collodion.

Absorbents and astringents have been employed to act upon the walls of the vessel. Until within very late years, good effects are said to have been obtained by the prolonged use of potassium iodide (10 : 200 a table-spoonful t. i. d.). This remedy should be particularly employed if we have reason to suspect that the formation of the aneurism is a result of syphilis. Less faith may be placed in the administration of acetate of lead (0.05 every two hours), or tannic acid (0.2 every two hours). Ergot and its preparations have been used considerably, and sometimes, it is said, with good effects.

Among the remedies which are said to give rise to the formation of clots within the aneurism, the galvanic current merits special mention. One or two needle-shaped electrodes are inserted into the aneurism, and the constant current allowed to pass through them for a certain length of time. According to Dujardin-Beaumetz (writing in 1877), Ciniselli and other Italian physicians had employed electro-puncture of aortic aneurisms forty-five times without seeing any bad effects from the operation. Among thirty-eight cases no result was obtained in eleven; in the remainder, improvement and recovery for a prolonged period were observed.

Anderson describes the following plan of application of the constant current:

The current should not be very strong, but possess considerable chemical power. Anderson employed, at the most, eight large elements of a Stöhrer battery. The needles should not be too thick, but very sharp. They should be insulated to within 2.5 cm. from the point. Before insertion, the needles should be oiled. One or both needles may be introduced, the latter particularly in large aneurisms. The positive pole produces a smaller but firmer clot than the negative pole. Each sitting should not last longer than an hour. According to its effect, the sitting is repeated at the end of one or more weeks. Recently it has been justly held that the anode alone produces a clot, and that therefore the cathode should not be inserted.

The injection of liquor ferri sesquichlorati has also been employed in order to give rise to the formation of a clot, but this plan is not without danger. The same object has also been sought by the introduction of foreign bodies into the aneurism. In two cases, Baccelli unsuccessfully inserted watch springs. Moore and Lewin introduced horse hair, Schroetter, fils de Florence (silk) into the aneurism.

The starvation plan of treatment possesses merely an historical interest.

## 2. *Stenosis and Occlusion of the Isthmus Aortæ.*

I. ANATOMICAL CHANGES.—The isthmus of the aorta is that portion situated between the origin of the left subclavian artery and the beginning of the descending thoracic aorta. During fetal life, this part is very narrow, and it is not until after birth that its lumen attains the size of the adjacent part of the aorta.

Under abnormal conditions, the isthmus may be stenosed or entirely occluded. These conditions are observed most frequently below the origin of the ductus Botalli, more rarely at the site of the latter, still more rarely above it (among forty-six cases, twenty-one immediately below, seventeen at the ductus, eight immediately above the ductus). Sometimes there is an annular constriction, sometimes a valve-like membrane juts internally from one side, sometimes the stenosis or occlusion extends over a distance of one centimetre. At the narrowed portion, the inner walls of the artery are not infrequently folded and furrowed, and may present thrombotic deposits.

In many cases the ductus Botalli presents no abnormality. It is occasionally found open. In rare cases it contained thrombi which projected into the lumen of the aorta.

The left ventricle is very often dilated and hypertrophied. The endocardium shows chronic inflammatory changes (thickening, calcification), most frequently at the aortic, but sometimes at the mitral valves. The disease may be associated, accordingly, with aortic insufficiency (six times among fifty-one cases collected by Duchek). Sommerbrodt states that there is a congenital deficiency in the number of aortic leaflets in twelve per cent of all the cases.

Very considerable dilatation is almost always found above the stenosis, *i. e.*, in the ascending aorta and arch. Aneurismal dilatation is also found occasionally immediately below the point of stenosis. The abdominal aorta, as a rule, is very small. The inner surface of the aorta often presents endarteritic changes (calcification, fatty degeneration).

As a matter of course, the supply of blood to the descending thoracic and the abdominal aorta would be interfered with to a dangerous degree

were it not aided by collateral channels. Branches of the subclavian artery in particular are extraordinarily dilated, and communicate with dilated and sinuous branches of the abdominal and thoracic aorta.

Stenosis or obliteration of the isthmus aortæ is associated not infrequently with other deformities, viz., openings in the cardiac septa, or other congenital heart lesions, hare-lip, club-foot, etc.

**II. SYMPTOMS AND DIAGNOSIS.**—The diagnosis of this condition depends on three symptoms:

1. The development of collateral circulation.
2. The character of the abdominal aortic pulse and the crural pulse.
3. The changes in the heart muscle.

The collateral circulation develops in order to supply the thoracic and abdominal aorta with blood. Branches of the subclavian artery, in part visceral, in part peripheral, take part in this process. The principal collateral branch among the visceral arteries is the inferior thyroid, which communicates with branches of the œsophageal and bronchial arteries. Among the peripheral branches the following are important: subclavian artery, internal mammary, anterior and superior intercostal, posterior intercostal, thoracic aorta.

Subclavian artery, transversa colli, dorsalis scapulæ, posterior intercostal, thoracic aorta.

Subclavian artery, transversa scapulæ, subscapular, posterior intercostal, thoracic aorta.

Subclavian artery, internal mammary, superior epigastric, inferior epigastric, iliac artery.

These collateral branches are unusually large and sinuous. They appear under the skin as vigorously pulsating vessels which attain almost the thickness of the little finger. In certain places, particularly near the inner border of the shoulder blade, they are aggregated so closely as to look like a cavernous tumor.

On palpation a thrill is often felt over the vessels. It coincides with the systole of the heart, but is a little later than the apex beat. These thrills may be so loud as to be perceptible to the patient as an annoying buzzing next to the sternum, along the ribs, or in the region of the back and scapula. Palpation is especially valuable in diagnosis if the dilatation is not sufficiently marked to be visible, but unusual pulsating movements are felt along the borders of the sternum, the ribs, and inner edge of the scapula.

A systolic murmur is usually heard on auscultation of the dilated vessels. In some cases there is an almost constant humming, which has been compared with the placental bruit. In one case, Leyden and Scheele described a systolic and diastolic murmur in the vessels.

The systolic murmur in the vessels is a little later than the apex beat of the heart. This is recognized best by ausculting the internal mammary artery near the right or left border of the sternum, when it will be found that the murmur follows immediately after the systolic sound of the heart. The murmurs are sometimes audible over the entire anterior abdominal wall.

The more marked the changes in the peripheral arteries the greater must be the stenosis of the isthmus aortæ.

The pulse of the abdominal aorta and the crural artery present two abnormal characteristics; in the first place, it is delayed in comparison with the apex beat and the radial pulse; and in the second place, it is extremely feeble, and may even be absent in the crural artery. The



slight filling and the retardation of the pulse are owing to the fact that the vessels receive their supply of blood by circuitous channels. The crural pulse is not alone delayed, but also assumes the tardy quality, as is shown especially well in sphygmographic tracings. The radial pulse, on the contrary, is unusually full, hard, and bounding, as the result of the dilatation and hypertrophy of the left ventricle.

These changes in the ventricle are the result of the increased obstacles offered by the stenosis or obliteration of the isthmus aortæ. But this symptom is not constant (twenty-five times in fifty-one cases according to Duchek). Dilatation of the ascending aorta and arch of the aorta is often recognized on palpation by pressing the finger deep into the jugular fossa.

Stenosis or obliteration of the isthmus of the aorta has been observed at all ages. Bochdalek described a case in a child of twenty-two days, and Raynaud observed it in an old man of ninety-two years. The lesion

FIG. 27.



Sphygmographic trace of retardation of the pulse between the radial and crural arteries in stenosis of the isthmus of the aorta. After Scheele.

is not very frequent. Hitherto a little more than seventy cases have been reported. The male sex is much more predisposed than the female.

In many cases the condition is very well tolerated and is only found accidentally at the autopsy. In other cases, the signs of cardiac insufficiency make their appearance. These often begin with palpitation, dyspnoea, cough, hæmoptysis, followed by œdema and other symptoms of stasis. The latter may be the cause of death.

In some cases death is the result of cerebral hemorrhage. Rupture of the heart or aorta, and sudden paralysis of the heart have been observed very often. Finally, death may result from intercurrent diseases, particularly pleurisy, pneumonia, and pericarditis.

III. ETIOLOGY.—In some cases the lesion seems to be a form of inhibited development, in which the isthmus retains its fetal calibre. Combination with other forms of inhibited development of the heart or other organs favors this view.

In other cases there seems to be an abnormal connection between the ductus Botalli and the aorta, the tissue of the former penetrating to a certain extent into the wall of the aorta. This renders it possible for the part of the aorta in question to take part in the post-fœtal processes of proliferation and obliteration of the ductus Botalli, and to undergo similar changes.

Hammernijk maintains that the process results from the proliferation into the aorta of a thrombus situated in the ductus Botalli. But this

is not true of all cases, since the aorta has been found stenosed, although the ductus was open. Moreover, closure of the ductus Botalli by thrombi is not a physiological process.

Finally, Lebert attributes the changes to foetal aortitis, but the cause of the latter condition is unknown.

IV. PROGNOSIS AND TREATMENT.—The prognosis is favorable in so far as the disease is compatible with a long lease of life.

The treatment depends on the principles laid down in the treatment of lesions of the heart. Bodily and mental excitement should be avoided. The diet should be light and nourishing. Symptoms of stasis or other complications must be treated according to the well-known rules.

### 3. *Congenital Narrowness of the Entire Aorta.*

Congenital narrowness of the entire aorta has been described a number of times. The lumen of the vessel in adults may not exceed that of the crural artery. The walls are often changed, the muscular coat and tunica intima being especially thin. These layers not infrequently undergo fatty degeneration and endarteritic changes, or the intima is wrinkled and full of folds. The aorta is generally very yielding and extensile, and the intercostal arteries are often given off irregularly.

These changes have been found particularly in chlorosis, combined frequently with defective development of the genital apparatus.

In rare cases this process constitutes an independent disease. Some patients are delicately built, others robust, but they are often pale, with slight powers of resistance, and subject to symptoms of stasis. The heart is generally hypertrophic and dilated, the peripheral pulses strikingly empty and small. Systolic murmurs may be heard, and are the result, perhaps, of irregular vibrations of the wall of the aorta. Pericardial friction murmurs are also heard occasionally. The disease ends, as a rule, with symptoms of increasing stasis or perhaps with rupture of the aorta. The diagnosis can be made only with a certain degree of probability.

### 4. *Rupture of the Aorta.*

Solution of continuity in the walls of the aorta may be the result of injury (knife wound, blow, fall from a height, etc.). In other cases the rupture follows changes in the walls of the vessel, particularly fatty degeneration of the intima and media, and endarteritic processes. The rupture is often preceded by aneurismal dilatation. It has also been observed in abnormal narrowness of the aorta, particularly when associated with considerable hypertrophy of the left ventricle.

In a third group of cases the rupture is the result of the extension to the aorta of ulcerative processes in the neighborhood, such as caries of the spine, cancer of the œsophagus, etc. This category also includes foreign bodies in the œsophagus.

Rupture of the aorta is more frequent in men than in women, and generally occurs at an advanced age. This is owing to the fact that men are more exposed to the exciting causes, and in addition changes in the walls of the aorta develop in later years.

The rupture may be entirely unexpected, or it may have been preceded by unusual bodily or mental exertion as the exciting cause.

If all the layers of the wall of the vessel are torn, the hemorrhage occurs into surrounding parts and death follows very quickly. The rupture is most frequent immediately above the origin of the aorta, so that hæmo-pericardium is produced. Rupture into the pulmonary artery or one of the cavities of the heart is rare. If the point of rupture is situated higher, the extravasation takes place into the posterior, rarely into the anterior, mediastinum. The pleura is sometimes separated from the thorax by the effused blood. In rupture of the abdominal aorta, the retro-peritoneal cellular tissue is infiltrated or, if the peritoneum is perforated, the blood escapes into the peritoneal cavity.

The rupture is usually transverse, and rarely exceeds two-thirds of the circumference of the aorta.

The rupture does not always extend through the entire thickness of the wall, but may involve only the intima and media, or the intima alone. If the intima and media are torn, the blood may undermine the adventitia, and separate it, for a considerable distance, from the tunica media (dissecting aneurism). The separation of the adventitia may extend from the origin of the aorta to the popliteal artery. In a number of cases, one or more openings, leading into the space between the outer layer and the two internal layers of the aorta, have been found in the lower part of the vessel, so that circulation within the dissecting aneurism was perfect. Externally, the dissecting aneurism appears as a spindle-shaped or sausage-shaped tumor. In many cases it delays complete rupture only for a short time, but Rokitsky showed that recovery does occur occasionally.

Cases in which the intima alone has ruptured, so that the blood flows between it and the media, are not frequent. In one case, Friedlaender found that the blood had ploughed its way between the outer and inner layers of the tunica media. The dissecting process sometimes extends to adjacent organs; for example, Cornil found blood between the layers of the pericardium.

Rupture of the aorta is shown, in some cases, by sudden and violent internal pains and many patients exclaim that something has burst internally. Then follow indescribable fear, rapidly increasing anæmia, rapid prostration, in short, the signs of internal hemorrhage. Areas of unusual dulness, affecting the heart, thorax, or abdomen, can often be demonstrated. Death occurs not infrequently within a few seconds. In other cases, the threatening symptoms subside, but reappear in a few hours or days, and terminate fatally. The dangerous condition very rarely terminates in a sort of recovery, but cases are known in which a dissecting aneurism has existed for years (professedly eleven years in a case reported by Goupil).



## SECTION II.

# DISEASES OF THE RESPIRATORY APPARATUS.

### PART I.

#### DISEASES OF THE NASAL CAVITY.

##### *Catarrh of the Nasal Mucous Membrane.* (*Catarrhal rhinitis. Coryza.*)

I. ETIOLOGY.—Catarrh of the nasal mucous membrane is usually the result of a cold, and is especially frequent in autumn and spring. It sometimes becomes almost epidemic. Certain individuals present a predisposition to nasal catarrh, while in others the effects of a cold are manifested in other organs.

Coryza is not infrequently the effect of local causes, such as injury, ulcerative processes, and eczema of the nasal mucous membrane, foreign bodies and new-growths in the nose.

The local causes also include the inhalation of dust, irritating gases, too cold or too warm air. For this reason, coryza occurs very often in certain trades, though the nasal mucous membrane may be gradually hardened to such irritants.

Many persons present an idiosyncrasy against certain odors. It is well known that certain individuals suffer from coryza on inhaling powdered ipecacuanha root.

This etiological group includes hay fever which results from inhalation of the pollen of graminacea. Toxic coryza includes that form which occurs after the administration of certain drugs, for example, iodine preparations. According to Stadion, digitalin also gives rise to coryza.

In some cases, coryza is a propagated inflammation (from abscess of the upper lip or the gums).

It appears often in acute and chronic infectious diseases (measles, scarlatina, variola, typhus, and typhoid fever, recurrent fever, whooping-cough, influenza, erysipelas, syphilis, phthisis, scrofula, leprosy, glanders, etc.).

In certain cases, there is a local infection of the nasal mucous membrane. Thus it is possible, though not observed with certainty, that the purulent secretion of gonorrhœa, when conveyed to the nasal mucous membrane, may give rise to a blenorrhœic inflammation. Fraenkel has explained some nasal catarrhs of the new-born by the entrance of the vaginal secretion of the mother into the nasal cavity of the child.

In some cases, the etiological connection is altogether obscure. Thus, certain women always suffer from coryza at the time of menstruation. I also know of

several cases in which men filling public offices always suffered, before appearing in public, from a rush of blood to the head, acute stoppage of the nasal passages and a discharge from the nose. This psychical coryza only ceased after they had been "in action" for a little while.

**II. SYMPTOMS.**—Coryza is more frequent in adults than in children. It may be either acute or chronic.

The acute form often begins with febrile symptoms. The patients suffer from repeated chilly sensations, feel extremely miserable and prostrated, complain of pains in the joints and limbs, and lose appetite. Then there is an increased thirst, and feeling of heat. The patients are often fearful of the onset of some serious disease, and, in very excitable individuals, delirium and even convulsions may be observed. These febrile prodromata occur most frequently when the disease is the result of a cold. They continue one to three days, rarely longer.

The first local changes are a feeling of dryness and a peculiar pricking sensation in the nose, which may increase to a slight sensation of pain. The nose is stopped up, speech becomes nasal, smell and generally taste are enfeebled or abolished, occasionally there are perverse sensations of smell; there is a constant tendency to sneeze.

A scanty, very thin, almost watery, clear secretion of a salty taste soon appears. According to Donders, it contains less chloride of sodium than chloride of ammonium. The latter substance is probably the cause of the irritating quality of the secretion which often gives rise to slight dermatitis of the upper lip. The secretion contains a few pus cells, and not infrequently ciliated epithelium.

After the catarrh has lasted for some days, the secretion gradually becomes more purulent. It grows richer in pus-cells and assumes a greenish, opaque appearance. At the same time the tendency to sneeze ceases.

The catarrhal changes are rarely confined to the nasal mucous membrane, but spread to adjacent cavities. Many patients complain of a sense of pressure above the root of the nose or of pain in the region of the eyebrows—the results of inflammation of the mucous membrane of the frontal sinus. The catarrh sometimes spreads along the lachrymal duct to the conjunctiva, and gives rise to epiphora, burning sensation in the eyes, dread of light, and redness of the conjunctiva. But the conjunctivitis sometimes precedes the nasal catarrh. If the inflammation spreads to the antrum of Highmore, the patients complain of pain in the cheek and along the alveolar border of the superior maxilla. If it spreads to the pharynx, they complain of a burning sensation in the pharynx and pain in deglutition. The mucous membrane of the Eustachian tubes may also be involved and give rise to partial deafness, pain, and ringing in the ears. Finally, if the catarrh spreads to the larynx, it causes cough and hoarseness.

If acute coryza of the new-born leads to stoppage of the nose, it may be followed by serious nutritive disturbances because the infant is accustomed to breathe through the nose while nursing. Death will occur from inanition, if nourishment is not administered in some other way. In addition, as the new-born breathe through the nose during sleep, the dorsum of the tongue being in contact, at the same time, with the hard palate, and as they do not know how to breathe through the mouth, stoppage of the nose readily leads to danger of suffocation and disturbs the sleep. It is readily understood that such conditions give rise to

pulmonary congestion which, in its turn, is followed by dyspnoea and cyanosis.

The course of acute coryza is almost always favorable and lasts only a few days. As a rule, it terminates at the end of the first week, more rarely it extends into the second week.

The infectious character of certain forms of coryza cannot be questioned. Thus, measles have been conveyed to healthy children by inoculation with the nasal secretion of patients suffering from measles. The rheumatic forms of coryza probably are also infectious.

Chronic catarrh of the nasal mucous membrane sometimes follows a frequently relapsing acute catarrh, or it runs a chronic course from the beginning. The latter form is found particularly in certain chronic infectious diseases (scrofula, syphilis). Febrile symptoms are absent unless there is a sudden exacerbation. The disease sometimes lasts a lifetime.

The patients complain usually of stoppage of the nose, so that they must breathe and sleep with the mouth open. Many persons acquire a stupid expression from this cause. Smell and taste are generally enfeebled, sometimes abolished. Speech is nasal. There is generally a profuse secretion of a yellow, purulent fluid, more rarely the fluid is thin and may amount to more than a pound in the twenty-four hours (rhinorrhœa).

Rhinoscopic examination shows that the mucous membrane is reddened, grayish-red, or brownish-red, covered with profuse secretion, and the nasal passages narrowed or closed on account of the swelling of the mucous membrane. The secretion very often dries into grayish-yellow or grayish-green crusts, which sometimes cover the mucous membrane like a mussel-like deposit, sometimes entirely occlude the nasal passages in certain places. They often give rise to the sensation of a foreign body and may be removed only with difficulty. They are sometimes converted into earthy concretions by the deposit of lime salts (rhinoliths).

The secretion sometimes undergoes putrid decomposition (ozæna simplex), and gives rise to an abominable stench. This is not alone disagreeable to those around them, but may produce in the patients an aversion to food. This condition is particularly apt to develop when the mucous membrane becomes atrophic (rhinitis chronica atrophica).

Ulcers sometimes form upon the mucous membrane, may spread deeply, and extend to the periosteum and bones. The bones then undergo necrotic changes. Such conditions are almost always associated with a stinking discharge (ozæna ulcerosa).

Chronic catarrh sometimes forms the basis for polypoid proliferations of the mucous membrane.

If the chronic inflammatory process spreads to the antrum of Highmore and the entrance to this cavity becomes occluded, an abundant accumulation of a usually mucoid fluid forms, dilates the cavity, and thins the bony walls (hydrops antri Highmori). This condition results occasionally in ulcerative processes. In the same way a very distressing chronic inflammation may develop in the frontal sinuses.

As a rule, the course of chronic nasal catarrh is benign, and the disease is annoying rather than dangerous.

Chronic catarrh and ozæna of the new-born are the result, in many cases, of congenital syphilis.



**TREATMENT.**—Prophylactic measures should first be considered. Delicate individuals should be hardened in a rational manner, and made more resistant to exposure. Residence at the sea-shore is specially indicated in many cases. The nasal mucous membrane should be protected from dust, irritating vapors, and the like.

Constitutional treatment must be adopted when the disease depends on syphilis, scrofula, or anæmia.

In addition, local treatment is often very useful. If the patient is suffering from an acute febrile coryza, it may be necessary to keep him in bed, put him on low diet, and administer an antipyretic. Diaphoresis is advisable in many cases (several cups of elder-tea, pilocarpin. muriat., 0.1 : 10, one syringeful subcutaneously, pulv. ipecacuanh. co., 0.5, etc.). The inhalation of warm vapors is agreeable to most patients. This is effected in the simplest manner, by directing the patient to place the face over a vessel of hot water, both being covered by a cloth. Solutions of salt, carbonate of soda, or sal ammoniac (0.5–1.0 [100]) may also be used with Siegle's inhalation apparatus. The attempts to abort acute coryza by means of applications of nitrate of silver or other astringents in solution are usually futile.

A great stir was occasioned some time ago by the following remedy proposed by Hagen, but it produces only temporary relief :

R Acid. carbolic .....	5.0
Spt. vini rectificat .....	15.0
Liq. ammon. caustici .....	5.0
Aq. destil .....	16.0
M. d. ad vitrum nigrum c. epistomate vitreo.	

S. A few drops poured on three to four sheets of thick paper and inhaled (with the eyes closed) every two hours.

If the upper lip is inflamed, it should be smeared with some oily preparation (vaseline, ol. cocois, ol. amygdalar.) three times a day.

In acute catarrh of the new-born, the nasal passages should be injected as often as possible with lukewarm water to prevent the formation of crusts.

The nasal douche and sternutatories must be resorted to in chronic catarrh of the nasal mucous membrane.

A douche of acid. carbolic. (2 : 200), or acetate of alumina (1 : 100) should be used three or four times a day, and followed immediately by a pinch of the following preparation :

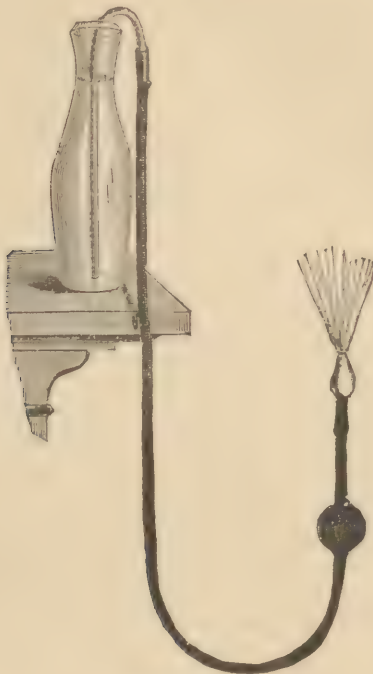
R Hydrarg. chlorid. mite .....	3.0
Alumini .....	5.0
M. D. S.	

Other authors recommend douches of solutions of alum, nitrate of silver, acetate of lead, sulphate of zinc, etc. Corrosive sublimate has also been used as a local application instead of calomel. Some writers prefer insufflations, tampons of cotton, brushing the mucous membrane, inhalations, spraying. The galvano-cautery has also been recommended.

A convenient form of nasal douche is shown in Fig. 23. It consists of a bent glass tube, which extends to the bottom of a litre vessel, and of a rubber tube with a bulb at the forward end, and an olive-shaped glass tube, which is placed in the nares. If the vessel is elevated, the bulb compressed, and then allowed to distend, the fluid will flow into the tube. During the application of the douche, the patient should keep the mouth open, so that the fluid entering one nostril can flow continuously out of the other. The nasal douche is preferable to an irriga-

tor, because the pressure of the fluid flowing from the latter may be too great, so that the fluid enters the Eustachian tube and the middle ear. The fluid used should always be lukewarm (28-30° R.), and  $\frac{1}{2}$ -1 litre may be employed at each sitting.

FIG. 28.



Nasal douche.

If crusts form, the nose should be injected with salt water or lukewarm water several times a day. In *ozæna simplex*, the treatment described above may be employed. Others prescribe solutions of hypermanganate of potash (1 : 100, one teaspoonful to one glass of water), chlorate of potash (10 : 200), thymol (0.1 : 200), chloride of calcium (5.0 : 200). Ulcers should be touched with lunar caustic or a concentrated solution of nitrate of silver (1 : 15). *Ozæna ulcerosa* and other complications require surgical treatment.

## PART II.

### DISEASES OF THE LARYNX.

#### 1. *Catarrh of the Laryngeal Mucous Membrane.* (*Laryngitis Catarrhalis.*)

I. ETIOLOGY.—Laryngeal catarrh is a very widespread disease. It occurs at every period of life, in men and women, and in all climates. But it is most frequent from the 20th to 40th years, and is also observed more often in men than in women. Rapid changes of temperature,

great moisture of the air, the prevalence of north and northeast winds, exposed situation of any locality, favor the development of the disease.

Among the special causes, colds occupy the first rank.

The noxious effects of the cold may be confined to the laryngeal mucous membrane, or also involve others (nose, conjunctiva, bronchi, etc.). Delicate individuals with a tendency to profuse perspiration are specially predisposed to the affection. A previous inflammation of the mucous membrane gives rise to a diminished power of resistance to subsequent rheumatic influences.

Laryngeal catarrhs are so frequent at certain times (particularly autumn and spring) that we are reminded of infectious influences.

The disease is produced not infrequently by noxa, which act directly on the laryngeal tissues. This is observed in the inhalation of irritating vapors (chlorine, ammonia, bromine, iodine, etc.), of dust (as in certain trades), in inveterate smokers, and after prolonged speaking, singing, shouting, or coughing (from over-exertion of the vocal cords).

Catarrh of the larynx is a frequent disease among clergymen, teachers, singers, officers. According to Labus, tenors are more liable to it than basses. In individuals suffering from diseases of the bronchi or lungs, hoarseness develops not infrequently as the result of laryngeal catarrh after the cough has been particularly violent and constant. This etiological group also includes those cases which develop in certain gastric affections, if eructations of rancid, irritating gases or the vomiting of acid masses have come in contact with the larynx, and given rise to inflammation. Even laryngeal ulceration may occur from this cause.

In certain cases the inflammation is conveyed from other parts. This occurs often in chronic diseases of the pharynx. For this reason it is observed often in drunkards, although several factors here come into play, viz., loud talking and singing, stopping in rooms full of smoke and draughts, etc.

Certain infectious diseases are associated commonly with laryngeal catarrh, for example, measles, influenza, hay fever, syphilis, etc.

Laryngeal catarrh is also very frequent in certain chronic constitutional diseases (Bright's disease, chlorosis, rachitis). Navratil maintains that diseases of the liver and spleen also predispose to catarrh of the larynx.

Some cases follow disturbances of circulation, as in thyroid enlargement and in heart disease. Finally, primary ulcerative processes or new growths inside of the larynx are attended, as a rule, with catarrh, at least of the adjacent parts of the mucous membrane.

Chronic inflammatory changes of the vocal cords develop not infrequently in boys at the age of puberty.

Mackenzie mentions some cases in which there was a widespread catarrhal predisposition of several mucous membranes, so that, for example, a man suffered from laryngitis, esophagitis, enteritis, and cystitis at the same time.

II. SYMPTOMS.—Acute laryngeal catarrh begins suddenly in the majority of cases. It begins rarely with general febrile symptoms (chill, elevation of temperature, increased frequency of the pulse, thirst). As a rule, the patients suffer from a feeling of tickling in the larynx and a desire to cough. There is sometimes a feeling of rawness in the laryngeal region, but pain is rarely noticed. The larynx is sensitive on pressure. Pain is sometimes produced by deglutition, either because the



epiglottis then presses on the inflamed parts, or because the latter are brought in contact with the food.

In the beginning the cough is dry. Later, the patient expectorates a transparent, vitreous, mucous sputum, which contains a few round cells, mingled with epithelioid cells; ciliated epithelium cells are rarely found. On the addition of acetic acid, the round cells swell, become transparent, and disclose one to three nuclei. At the same time, mucin (the chief constituent of the sputum) is deposited in the form of veil-like clouds. As the catarrh subsides, the sputum becomes muco-purulent. It becomes more profuse, thinner, richer in water and poorer in mucin, and contains a much larger number of round cells.

The sputum sometimes contains bloody streaks or dots, particularly if the cough is very severe and the inflammation intense. A larger admixture of blood is very rare.

The cough sometimes becomes spasmodic in character. It occurs in spells, and is associated with loud inspiratory stridor, the result of spasmodic occlusion of the glottis.

The voice is very often affected. It becomes indistinct, muffled, often breaks into falsetto, finally becomes aphonic, and sinks into a gentle whisper. In some cases the laryngoscope shows marked swelling and irregularities on the vocal cords, which prevent an accurate adjustment of their free edges; in others some of the muscles of the vocal cords are paretic, or parts of the mucous membrane, which are adjacent to the vocal cords, are swollen and inserted between the vocal cords. Moreover, the false vocal cords may be swollen to such an extent that they project in great part over the true vocal cords, and act as a damper.

The diagnosis of laryngeal catarrh cannot be made with certainty from the vocal disturbance. A positive diagnosis can only be made with the aid of the laryngoscope. As a rule, this shows a circumscribed, more rarely a diffuse catarrh.

Acute catarrh of the true vocal cords is recognized by their change in color from a glossy tendon-white to a bright red. At the onset of the disease, the hyperamic vessels are recognized not infrequently as fine red streaks. As the catarrh progresses the redness becomes diffuse, and the vocal cords assume a flesh color. They are sometimes thickened and uneven, particularly at their free edges. At first they have a peculiar dry appearance, and have but little gloss; later, their surface is very moist, and not infrequently covered in part with mucous or muco-purulent fluid. The fluid is apt to accumulate between the free edges of the vocal cords, so that, when the latter are separated during phonation or deep inspiration, the fluid gathers in tough, long threads between them. The catarrh is not always distributed uniformly over the vocal cords, but is apt to be localized in those parts which cover the apices of the arytenoid cartilages, more rarely near the anterior insertion of the cords. The catarrh may be confined to one vocal cord.

If the catarrh is associated with paralysis or paresis of some of the laryngeal muscles—the result of so-called myochorditis, from inflammatory serous infiltration of the muscles—this is recognized by the abnormal position of the cords during phonation or respiration. The internal thyro-arytenoid and arytenoid muscles are most apt to be affected.

In acute catarrh of the false vocal cords, the surface of these parts is found to be unusually red, swollen, and moist. If the swelling is considerable, the parts may project to such an extent into the cavity of the larynx as to cover the true vocal cords entirely, or almost entirely.

In attempts at phonation, the free edges of the true vocal cords are alone visible (Fig. 29).

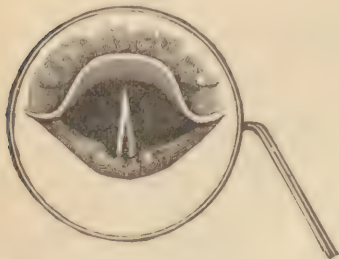
Inflammation of the mucous membrane of the arytaenoid cartilages and epiglottis presents corresponding appearances.

In rare cases, the inflamed mucous membrane contains extravasations of blood in the form of streaks and patches, which are partly isolated, partly confluent (laryngitis hæmorrhagica). In one case, Tobold recognized the bleeding vessel with the laryngoscope. Laryngitis hæmorrhagica should not be mistaken for the blood which remains inside of the larynx after hæmoptysis or hæmatemesis.

The duration of acute laryngeal catarrh is often only one to two days; occasionally it lasts one to two weeks, rarely longer.

Serious complications are infrequent. In some cases, however, the

FIG. 29.



Laryngoscopic image of laryngitis with marked swelling of the false vocal cords.

FIG. 30.



Laryngoscopic image in laryngitis hæmorrhagica (the darker spots are extravasations of blood).

swelling of the mucous membrane is so excessive that symptoms of laryngeal stenosis develop and are followed by death from suffocation (catarrhus laryngis acutissimus).

Temporary symptoms of laryngeal stenosis are not infrequent in childhood (pseudo-croup). The stenotic symptoms usually develop suddenly at night, having been preceded during the day by catarrhal symptoms on the part of the conjunctiva, nasal and laryngeal mucous membranes. After sleeping quietly during the first part of the night, the children often start out of a sound sleep, crying that they are choking. They toss to and fro in bed, sit up and grasp the throat in which the obstruction to respiration is situated. At the same time they present the signs of intense inspiratory dyspnoea. The auxiliary muscles of respiration are brought into play, inspiratory retraction of the thorax takes place, inspiration is slow and accompanied by a loud, stridulous sound, the face grows livid, and the features express evident fear of death. The little patients are hoarse, not infrequently entirely aphonic, the cough is hoarse and barking. This scene continues for one or more hours, and ends spontaneously or after the administration of an emetic. Relapses are frequent.

The causal factors of pseudo-croup are not always alike. In some cases it is the result of an accumulation of mucus which collects, during sleep, above the rima glottidis, and causes the free edges of the vocal cords to adhere to one another. Ziemssen attributes some cases to spasm of the glottis. In one case, I noticed such marked swelling of the false vocal cords that they came in contact with one another during inspiration. In other cases, I found marked swelling and respiratory im-

mobility of the true vocal cords, and very pronounced swelling of the mucous membrane in the inter-arytenoid fossa, so that the latter projected into the rima glottidis.

Chronic laryngeal catarrh either appears as such at the start, or it develops from neglected or frequently recurring acute catarrh. The inflammation is generally chronic from the beginning when the disease is the result of chronic constitutional anomalies, impairment of circulation, continued over-exertion or mechanical irritation of the vocal cords.

The subjective symptoms are usually slighter than in acute catarrh, so that some patients complain very little, or not at all, of the sensation of tickling and the desire to cough. Febrile movement is absent, unless some complication is present.

The sputum is similar to that of acute catarrh. The voice suffers in the same way as in the acute form, but the vocal symptoms are sometimes not noticeable, for a brief period, if the patient talks in a loud tone.

The laryngoscope shows redness, swelling, and increased secretion. But the redness is not so bright as in acute laryngitis; it is brownish or grayish-red, sometimes even bluish-red. The remains of previous hemorrhages appear as brownish or blackish spots, and some of the vessels are dilated and sinuous.

The enlargement of the laryngeal structures may attain considerable dimensions. The true vocal cords may increase twofold or threefold in size, or the epiglottis may be converted into a shapeless mass. Localized villous enlargements of the mucous membrane are sometimes noticed. If the swelling affects chiefly the mucous follicles, the larynx assumes a finely nodular or granular appearance (*laryngitis granulosa*). In some cases, the hypersecretion results in a sort of blennorrhœa of the mucous membrane of the larynx.

Ulcers sometimes form as the result of chronic catarrh. They begin as slight epithelial erosions, and then spread deeper, or they develop from ulceration of the mucous follicles. They are most frequent at the free edges of the true vocal cords. Their development here is probably favored by the approximation and friction of the vocal cords against one another during phonation. Catarrhal ulcers develop with special frequency at the apex of the arytenoid cartilages. But ulcers are rarely an outcome of catarrh from cold; they are usually the result of infectious influences (phthisis, syphilis, etc.). The ulcers at the free edges of the vocal cords sometimes assume a slit-shaped appearance.

In certain cases, chronic catarrh of the larynx gives rise to the development of tumors (polypi or papillomata).

A very serious complication is known as *chorditis vocalis hypertrophica inferior*. This is an inflammatory hyperplastic new-formation and proliferation, which starts from the mucous membrane and connective tissue of the lower surface of the vocal cords, and projects beneath them, like a diaphragm, into the cavity of the larynx (Fig. 31). These changes prove dangerous by producing laryngeal stenosis, which is often the cause of death.

After long-standing catarrh, the vocal cords sometimes assume a warty, granular condition (*chorditis tuberosa*, s. *trachomatosa*).

III. ANATOMICAL CHANGES.—We have nothing to add to the description of the laryngoscopic appearances mentioned in the preceding section. Indeed, laryngoscopic examination is preferable to post-mortem examination, inasmuch as the laryngeal mucous membrane is very rich



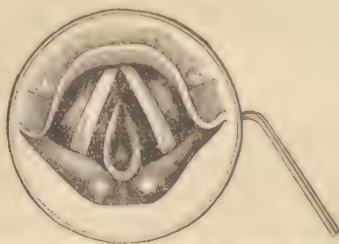
in elastic fibres, so that hyperæmia and swelling of the parts diminish after death.

The microscopical changes are the well-known ones of inflammation: dilatation of the blood-vessels in the mucosa and submucosa, swelling of these parts from serous transudation, emigration of white blood-globules, proliferation of the connective-tissue cells and epithelium, partial desquamation of the latter. In chondritis hypertrophica, Ganghofer and Chiari found the same histological changes as in rhinoscleroma.

IV. DIAGNOSIS.—The laryngoscope at once discloses the situation, extent, complication, and nature of the catarrh. But it must not be forgotten that the anatomical changes and functional disturbances do not always correspond, and that sometimes severe symptoms follow slight anatomical changes and vice versa.

The laryngoscopic examination is especially valuable in the differential diagnosis of croup and pseudo-croup, as the latter never presents fibrinous deposits within the larynx. It must be remembered, however,

FIG. 31.



Laryngoscopic image in chondritis hypertrophica inferior. After E. Burow.

that the use of the laryngoscope in children is attended with great technical difficulties.

V. PROGNOSIS.—Although laryngeal catarrh rarely endangers life, the prognosis as regards complete recovery is not always favorable. It depends chiefly on the etiology, and permanent benefit can be hoped for only when the cause can be removed.

VI. TREATMENT.—Prophylaxis is very important in the treatment of the disease. Delicate individuals must be hardened by the use of cold rubbings and douches, suitable clothing, rational exercise in the open air, a sojourn at the sea-shore. Persons working in dusty rooms must take suitable precautions against the inhalation of dust.

Acute laryngeal catarrh following a cold is often relieved in a few days by diaphoretic measures.

The patient is kept in a room at a temperature of 15° R., or, if fever is present, is kept in bed and a copious quantity of warm teas given to him.

If the sensation of tickling in the larynx or the desire to cough is very pronounced, we may give small doses of narcotics: morphin. muriat. (0.002 every 2 hours), pulv. ipecacuanh. opiat. (0.05 every 2 hours); aq. amygdalar. amar., 10.0; morphin. muriat., 0.1. M. D. S., ten drops on coughing. I have very often seen good effects from troches of potassium bromide and morphine (kali brom., 1.0; morphin. muriat., 0.02; tragacanth. et sacch. alb., q. s. ut f. trochisci No. x. D. S., a troche every hour or two).

If the expectoration is very scanty and tough, inhalations of alkalies may be employed to loosen the secretion.

This is done preferably by means of Siegle's inhalation apparatus. The vessel should only be half full of water in order to prevent an explosion. The glass vessel at the front is filled with the fluid to be in-

FIG. 32.

Siegle's inhalation apparatus.  $\frac{1}{2}$  natural size.

haled. The opening of the tube conveying the vapor should be on a level with the patient's mouth. The patient draws the tongue out of the mouth by means of the thumb and forefinger which are covered with a cloth. The narrow opening of the funnel is placed on the dorsum of the tongue, and the vapor is inspired as deeply as possible. The inhalations are repeated every two to three hours. It is also advisable to impregnate the atmosphere of the room every two to three hours with vapor from the apparatus. The following solutions may be used for inhalation:

Sol. natrii chlorati,	. . . . .	1.0-5.0 : 100
Sol. natrii bicarbonic.,	. . . . .	1.0-5.0 : 100
Sol. natrii bicarbonic.,	. . . . .	1.0-3.0 : 100
Sol. ammonii muriatici,	. . . . .	1.0-3.0 : 100
Sol. kalii bromati,	. . . . .	1.0-3.0 : 100

Ems water may also be used for inhalation. Ems or Selters water, mixed with an equal amount of hot milk, may be taken every morning.

If the expectoration has become profuse, the following preparations may be used:

Sol. argent. nitric.,	. . . . .	0.1-1.0 : 100
Sol. acid. tannic.,	. . . . .	0.1-3.0 : 100
Sol. alumin.,	. . . . .	1.0-3.0 : 100
Sol. alumin. acetic.,	. . . . .	0.3-1.0 : 100
Sol. zinc. sulphuric.,	. . . . .	0.1-0.5 : 100
Sol. zinc. chlorati,	. . . . .	0.3-1.5 : 100
Sol. ferri sesquichlorat.,	. . . . .	0.03-0.3 : 100

We should always begin with weak solutions and gradually increase their strength. The preparation should be changed every three to four days, in order that the mucous membrane may not become accustomed to its use.

The abortive treatment of acute laryngeal catarrh is not entirely devoid of danger. In some cases surprisingly rapid results are obtained, in others the inflammatory process is intensified. The interior of the larynx is brushed with a strong solution of nitrate of silver (1-3 : 30),

FIG. 33.

Laryngeal brush.  $\frac{1}{2}$  natural size.

FIG. 34.

Laryngeal sponge.  $\frac{1}{2}$  natural size.

which is introduced either with the aid of a laryngoscope or as best we can with the patient's mouth wide open and tongue protruding. A brush (Fig. 33) or sponge (Fig. 34) may be employed. The former is preferable because it is more readily cleaned.

Inhalations of astringents are also indicated in hemorrhagic laryngitis. Concentrated irritant solutions should be avoided. *Liquor ferri sesquichlorati* (0.1-0.3 : 100) is particularly indicated.



Catarrhus acutissimus and pseudo-croup are treated in part in the same way. Emetics should be administered and congestion of the skin of the laryngeal region produced by the application of sponges dipped in hot water, by alcoholic rubbings, sinapisms, or blisters. Three to six leeches may be applied to the region of the larynx or, in children, to the manubrium sterni, warm water should be ingested copiously, and tracheotomy performed if death from suffocation is imminent. In many cases of pseudo-croup, benefit is derived from the plan recommended by Niemeyer, viz., that the children should not be allowed to sleep too soundly, but should be awakened several times during the night and allowed to drink hot tea.

Briandt maintains that some cases of pseudo-croup are the result of malarial infection, and are rapidly relieved by quinine.

The treatment of chronic laryngeal catarrh relies in great part on the measures just recommended. The etiology must also be considered. In syphilitic individuals, anti-syphilitic remedies are indicated; likewise inhalations of corrosive sublimate (0.1-0.2 : 100), or iodide of potassium. Appropriate constitutional treatment must also be adopted in chlorotic and anæmic, and in rachitic and scrofulous individuals. When the disease is associated with chronic catarrh of the pharynx, this must first be relieved. Many cases are associated with hyperplasia of the uvula, the lower end of which touches the entrance to the larynx. In such cases the surgical shortening of the uvula has been observed to have a surprising effect on the secondary laryngeal catarrh.

If the patients are puffy and plethoric, we should prescribe cures of Carlsbad, Marienbad, Kissingen, Hamburg, Tarasp, and similar waters, or should carry out laxative treatment at home.

Alkaline muriatic waters, sool baths, sulphur waters enjoy great repute in the treatment of this disease.

In many cases the results of treatment are but temporary, and the catarrh soon returns when the patients resume their ordinary occupations. The outlook is even worse when the patient cannot be withdrawn from the injurious influences which produced the disease. In such cases we may also employ additional therapeutic measures. One of these is the use of hydropathic applications to the neck. A napkin is dipped in cold water, then compressed, placed around the throat, and covered with a dry cloth. This compress is kept on over night.

Derivatives to the laryngeal region are also employed, for example, painting with tincture of iodine, inunctions with ol. terebinth., ol. crotonis, ãã 3.0, three drops applied every night until pustules form, ung. stibio-kali tartarici, a piece as large as a pea rubbed in every night until pustules form.

In a few very obstinate cases, I have used compressed air with very rapid and excellent success. Faradization of the larynx may also prove effective.

Ulcerations should be cauterized. The button-shaped end of a flexible wire is heated, brought in contact with lunar caustic, allowed to cool, and then applied, with the aid of the laryngoscope, to the surface of the ulcer.

Cauterization, scarification, and dilatation with sounds have been successfully adopted in chondritis vocalis hypertrophica inferior; in other cases tracheotomy was rendered necessary. Good effects have also been obtained from the internal administration of potassium iodide, probably because there was a syphilitic element in the case.

Labus has obtained good results from the use of the curette in other hyperplastic changes of the mucous membrane of the larynx.

## 2. *Edema of the Glottis. Angina laryngea submucosa.*

I. ANATOMICAL CHANGES.—The term *angina laryngea submucosa* refers to all stenoses of the laryngeal cavity resulting from changes in the submucous connective tissue. Sometimes we have to deal with purely oedematous conditions (*oedema glottidis*), sometimes with serous accumulations from previous or coincident inflammation (*laryngitis phlegmonosa* s. *submucosa*), sometimes with an accumulation of sero-purulent fluid (*laryngitis submucosa sero-purulenta*), and finally with circumscribed depots of pus (*abscessus laryngis*).

Swelling occurs most frequently and to the most marked extent on the posterior surface of the epiglottis and in the ary-epiglottic folds, because the connective tissue of the submucosa in these localities is particularly loose and large meshed. The epiglottis is then converted into a shapeless mass looking like a thumb, and the ary-epiglottic folds may attain the dimensions of a pigeon's egg. Michel has recently called attention to oedematous swellings on the anterior surface of the epiglottis (*angina epiglottica anterior*).

Swelling of the arytenoid cartilages and false vocal cords is next in frequency. Further down the submucous connective tissue is so firm that any considerable accumulation of fluid is usually no longer possible. Massei has reported a case in which there was oedematous swelling of the submucosa covering the true vocal cords, so that the mucous membrane looked like two bladders projecting into the rima glottidis. In rare cases oedematous swelling has also been found below the vocal cords. These changes are generally bilateral. They are unilateral only when dependent on local causes (ulceration, unilateral compression of veins, etc.). This is more frequent on the right side, and, indeed, the changes here are generally more marked than on the left side.

The affected parts are particularly noticeable on account of their intumescence. The mucous membrane is sometimes reddened, sometimes extremely pale. The latter appearance may be presented even in cases which follow inflammatory processes.

On making an incision, clear serous fluid escapes in many cases, the swelling disappears, and the mucous membrane becomes wrinkled. In other cases only a few drops escape, and a gelatinous substance remains in the submucosa. In other cases the fluid is a little cloudy and flocculent (sero-pus). Purulent infiltration of the submucosa or a gangrenous condition of the infiltration is very rare. Circumscribed depots of pus or hemorrhagic extravasations are also infrequent. If the oedema has lasted a long time, hyperplastic processes are observed not infrequently in the mucosa, submucosa, and perichondrium.

Every stenosis of the laryngeal cavity must interfere with respiration, and if it gets the upper hand, will lead to death from suffocation. Lisfranc shows that in bodies in which there was oedematous swelling of the epiglottis and ary-epiglottic ligaments, these parts were pressed against one another and occluded the entrance to the larynx when an attempt was made to blow air from the mouth into the larynx. On the other hand, the air passed readily upwards from the trachea. Hence, under such conditions, inspiration is chiefly interfered with.

II. ETIOLOGY.—Oedema of the glottis develops most frequently after

previous diseases of the larynx. Mechanical, chemical, and thermal irritation of the laryngeal mucous membrane may also give rise to oedema glottidis. It has been observed after swallowing sharp foreign bodies, which pricked the laryngeal mucous membrane, and after the ingestion of mineral acids or caustic alkalies. It may also follow the inhalation of hot vapors. This form occurs with relative frequency in children in whom oedema glottidis is otherwise extremely rare. (Oedema glottidis is not infrequent after ulcerative processes in the larynx (catarrhal, tubercular, syphilitic, typhoid, carcinomatous, perichondritic ulcers). Injuries to the larynx, whether the result of surgical operations or not, occasionally give rise to this affection. All the forms hitherto mentioned belong to the category of inflammatory oedemas.

The disease also occurs as the result of inflammation propagated from adjacent parts (angina, glossitis, parotitis, inflammation of the connective tissue of the neck, etc.).

Oedema of the glottis may complicate certain infectious diseases (erysipelas, typhoid fever, small-pox, measles, scarlatina, diphtheria, glanders, pyæmia, whooping cough). In such cases, also, we have to deal with inflammatory, probably metastatic-inflammatory oedema. In cases of small pox, the eruption is found not infrequently upon the laryngeal mucous membrane. It is noteworthy that in certain epidemics of the diseases mentioned, the complication with oedema glottidis is a frequent one. Boeckel observed a complication with oedema glottidis in a case of *ecthyma*.

Stasis oedema or hydramic oedema of the glottis develops when the conditions necessary to the development of general or local dropsy are furnished. It is observed, though not frequently, in Bright's disease and in chronic pulmonary and cardiac affections. Fauvel and Waldenburg maintain that dropsical swelling of the laryngeal mucous membrane is sometimes the first manifest symptom of nephritis. (Oedema glottidis also develops in cachectic conditions (malaria, syphilis, cancer, waxy degeneration, convalescence from severe diseases).)

Among the local causes of stasis are tumors which prevent the flow of blood from the laryngeal veins: goitre, enlargement of lymphatic glands, mediastinal tumors, aortic aneurisms, etc.

Finally, the origin of some cases is unknown. Marboux and Hecht mention the case of a patient who, while bathing, was plunged under the water; this was followed by oedema glottidis. Idiopathic abscesses of the larynx have also been reported.

The disease is more frequent in men than in women (70 per cent in the male sex, according to Sestier). It is very rare in children, most frequent from the ages of 18 to 50 years. Among 149 cases collected by Sestier, only 17 occurred before the age of 15.

III. SYMPTOMS.—The symptoms sometimes develop so suddenly, and reach a dangerous height so quickly, that life is extinct before medical aid can be secured. In another series of cases, the symptoms develop more slowly, but they uninterruptedly assume a more and more threatening character. Finally, the disease may last for weeks, and present exacerbations and remissions.

The symptoms are those of laryngeal stenosis: inspiratory dyspnoea, inspiratory retraction of the thorax, sibilant and stridulous inspiration, often hoarse voice, difficulty in deglutition, barking cough, and not infrequently attacks of suffocation.

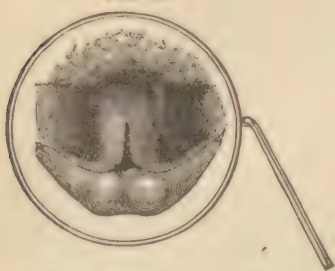


The situation and extent of the obstruction are determined by internal palpation and laryngoscopic examination.

In performing internal palpation, the mouth should be kept wide open and the tongue protruded as far as possible. The physician should grasp the tongue between the thumb and forefinger of the left hand (which has been covered with a cloth), the index finger of the right hand is introduced into the mouth and rapidly pushed deep along the root of the tongue. If the epiglottis is œdematous, this part is felt as a swollen, sausage-shaped body, and the thickened ary-epiglottic folds are also readily felt. The finger cannot be pushed beyond these parts.

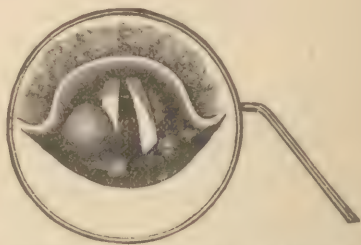
The swollen epiglottis is sometimes visible from the mouth, if the tongue is pressed vigorously downwards and forwards with the aid of a spatula. Voltolini recommends the following procedure. The tongue is grasped between the thumb and index finger of the left hand, and the third and fourth fingers of the same hand are placed on the sides of the pomum Adami. The tongue is then drawn forwards and downwards,

FIG. 35.



Laryngeal image in diffuse œdema of the glottis.

FIG. 36.



Laryngeal image in œdema of the mucous membrane of the arytenoid cartilages on the right side.

while the Adam's apple is vigorously lifted upwards with the aid of the third and fourth fingers. If the root of the tongue is depressed with a spatula, the epiglottis becomes clearly visible.

Laryngoscopic examination is not always easily effected, on account of the fear of suffocation from which the patients suffer. In œdema of the epiglottis and ary-epiglottic folds, the larynx is found surrounded by swollen ridges (Fig. 35), in local œdema there are circumscribed prominences (Fig. 36). These yield on pressure with the laryngeal sound, and give rise to pain if an ulcer or abscess is situated beneath them.

The subjective complaints consist, in the main, of the fear of suffocation. The patients complain, in rare cases, of a sticking pain in the larynx, or a feeling as if a foreign body were lodged there. When symptoms of carbonic-acid poisoning appear, the face assumes a leaden gray color, the sensorium is clouded, twitchings occur in the limbs, and death supervenes.

**IV. DIAGNOSIS.**—The diagnosis admits of no doubt when palpation or laryngoscopic examination furnishes positive results. If reliance is placed on the clinical symptoms alone, we must also exclude other causes of laryngeal stenosis, particularly croup, pseudo-croup, polypi, foreign bodies, and retro-pharyngeal abscess.

**V. PROGNOSIS.**—The prognosis is very grave. Among 213 cases collected by Sestier, 158 proved fatal.

The more acute the development of the symptoms of stenosis, the more serious is the course of the affection. The prognosis also depends upon the primary causes of the disease, since some of these maintain a tendency to relapse.

VI. TREATMENT.—As a rule, the question of treatment does not arise until the symptoms of stenosis make their appearance. As a general thing, very little can be expected from internal remedies.

When the changes were of an inflammatory character, Niemeyer observed good results from the slow ingestion of pieces of ice. The application of iced compresses to the throat may also be tried. Sinapisms, vesicants, local and general blood-letting, may be employed only when there is no immediate danger to life. Some authors have reported good results from applications of nitrate of silver (1 : 10) to the laryngeal mucous membrane.

Vigorous cathartics have been known to have a favorable action. They seem to us to be particularly indicated in hydræmic œdema (Inf. sennæ co. 180.0, natri sulphuric. 20.0. M. D. S. One tablespoonful every two hours. Inf. colocynth. 2 : 180.0, syr. sennæ 20.0. M. D. S. One tablespoonful every two hours. Ol. crotonis 0.05, ol. ricini 30.0, gummi arabic. 7.5, f. c. aq. destil. q. s. emulsio 180.0, syr. sennæ 20.0. M. D. S. One tablespoonful every two hours).

In many cases scarification may be performed (with the aid of the laryngoscope) with a curved bistoury which is covered with adhesive plaster as far as the tip. This plan is especially indicated in laryngeal abscess. In œdema of the epiglottis and ary-epiglottic ligaments, it has been recommended to tear the mucous membrane with the sharpened nail of the right index finger. In abscess of the larynx, early intralaryngeal incisions should be made. Sometimes the abscess attempts to escape externally, and must be opened through the skin. A few cases of spontaneous rupture and recovery have been reported. \*

If a decided effect is not produced, tracheotomy should be performed. Artificial respiration may be necessary after the operation has been performed.

The operation should not be delayed too long. In urgent cases the simplest instruments, even a pen-knife, will suffice. Many a patient has lost his life while the physician was waiting for his case of instruments. A patient suffering from œdema glottidis should always be under medical supervision, since tracheotomy may become necessary at any moment.

A catheter has sometimes been introduced between the vocal cords in order to render possible the entrance of air (so-called tubage).

### 3. *Inflammation of the Perichondrium of the Cartilages of the Larynx.* *Perichondritis Laryngea.*

I. ETIOLOGY.—Perichondritis laryngea rarely develops as an independent, primary disease. Injury (with or without fracture of the cartilages), cold, and even over-exertion of the vocal cords from loud singing or speaking, are mentioned as causes.

It is said that the ossification of the cartilages of the larynx in old age may give rise to perichondritis.

As a rule, the disease is secondary to inflammatory and ulcerative processes of the laryngeal mucous membrane (catarrhal, tubercular, syphilitic, cancerous ulcerations, and those following typhoid fever, small-pox, pyæmia, and other infectious diseases).

The disease is more frequent in males than in females. It is most frequent from the age of twenty to forty years.

II. ANATOMICAL CHANGES.—Perichondritis may attack any of the laryngeal cartilages, so that we differentiate perichondritis arytenoidea, cricoidea, thyroidea, and epiglottidea. This also expresses the order of frequency with which the different cartilages are affected. Not infrequently the inflammation spreads from one cartilage to another.

As the result of the inflammation, pus accumulates between the cartilage and the perichondrium, separating them from each other. The cartilage may be situated, under certain circumstances, in a pus cavity. This may attain considerable dimensions; for example, the arytenoid cartilages have been known to attain the size of a large cherry, and in other places the abscesses have grown as large as a small egg. The pus is often thin and ichorous, particularly after perforation has occurred. The stage of abscess formation is attended with great danger, because the laryngeal space is thereby narrowed. Moreover, the abscess is often complicated with œdema of the submucosa, which adds to the stenosis.

These changes affect the nutrition of the cartilage and perichondrium. As the perichondrium maintains the nutrition of the cartilage, the latter must undergo necrosis when it is separated from the former. The necrotic parts are cast off, and, if the abscess ruptures, are often expectorated. The pieces of cartilage are blackish, porous, partially calcified, or fibrous and horn-like, or fatty and gelatinous. Their size varies, but sometimes their origin can be recognized from the shape.

The perichondrium may also be destroyed. The walls of the abscess often form a thick new membrane whose inner surface is covered with a thick, cheesy layer of pus.

If the abscess has ruptured and the cartilage has been discharged, the development of cicatricial tissue may give rise to a sort of recovery, though, secondary laryngeal stenosis may finally result. In some cases proliferations of the cartilage or perichondrium occur, and may undergo ossification. If large pieces of cartilage have been exfoliated, the larynx may suddenly sink in, and thus give rise to occlusion of its lumen, and sudden death from suffocation.

Arytenoid perichondritis occurs most frequently when ulcers have formed upon the posterior end of the true vocal cords, and particularly on the processus vocalis. A very small fistulous opening sometimes leads into the abscess cavity.

Cricoid perichondritis affects usually the posterior thickened part of the cartilage. Dittrich explained its occurrence in typhoid fever as the result of pressure of the cricoid plate against the anterior surface of the spine, especially as similar changes were found not infrequently on the opposite surface of the œsophagus. Ziemssen maintains that frequent introduction of the œsophageal sound in old people with ossified and thickened cricoid cartilages may produce perichondritis in a mechanical manner. When the abscess breaks, the pus enters the larynx or the beginning of the œsophagus. The perforation may also take place into both localities at the same time, thus giving rise to a laryngo-œsophageal fistula. The pus may also burrow along the side of the neck, forming long, sinuous fistulæ.

In thyroid perichondritis the pus may rupture into the cavity of the larynx, or externally through the integument. If it ruptures in both directions, a laryngeal fistula is formed through which fluids may be injected into the larynx.



Epiglottic perichondritis is extremely rare, and, as a rule, occurs only when inflammatory processes are present in some of the other cartilages.

III. SYMPTOMS.—The symptoms are defined most clearly when the disease is primary. The secondary form is preceded for a longer or shorter time by other functional disturbances. As a rule, primary perichondritis presents a tendency to an acute, secondary perichondritis to a chronic course.

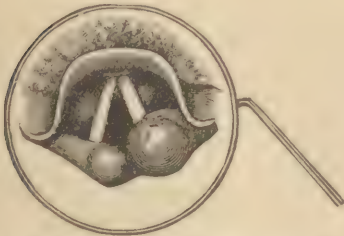
Pain in the larynx is a prominent symptom. Sometimes it occurs spontaneously as a burning or sticking sensation inside of the larynx, sometimes it is produced by pressure on the larynx, or by the act of deglutition. Strict localization of the pain on pressure indicates, with a certain degree of positiveness, the situation of the perichondritis. Pain in deglutition is produced when the arytenoid cartilages, or the posterior plate of the cricoid cartilage, are the site of the disease.

Difficulty in deglutition is not alone the result of the pain, but may also be produced mechanically if an abscess forms on the posterior surface of the larynx, projects into the lumen of the œsophagus and occludes it. It sometimes become necessary to maintain the patient's nutrition with the aid of the œsophageal sound or nutritive enemata. It is unnecessary to say that the introduction of the sound does not exert a favorable effect upon the inflammation.

Swelling, redness, and doughy œdema of the integument of the neck sometimes point to an abscess beneath the perichondrium of the thyroid cartilage. Enlargement of the cervical lymphatic glands has been repeatedly observed.

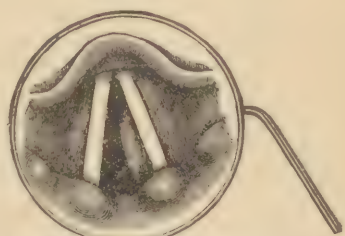
Disturbances of phonation are almost always present. In a series of cases they are the result of the primary disease, in others, of mechanical obstruction to the mobility of the vocal cords. In a third series of cases

FIG. 37.



Laryngeal image in left arytenoid perichondritis.

FIG. 38.



Laryngeal image in left lateral cricoid perichondritis. After Ziemssen.

the muscular apparatus of the vocal cords is affected by secondary inflammation.

Respiratory disturbances (signs of laryngeal stenosis and inspiratory dyspnoea) are very often observed. The swelling in itself is often sufficient to explain these symptoms. They may rapidly attain a dangerous height if the abscess becomes complicated with œdema of the submucous tissue. In some cases paralysis of the dilators of the vocal cords (crico-arytenoidei postici) may give rise to or intensify the inspiratory dyspnoea.

The laryngoscopic appearances are very important. Abscesses are recognized by the tumefaction, though small ones occasionally remain

concealed. The swelling is found to fluctuate on contact with the sound.

The course of perichondritis laryngea is attended with great dangers. Sudden death from suffocation may occur as the result of the abscess or of oedema glottidis. In some cases the symptoms of suffocation disappear rapidly after the expectoration of masses of pus, which are often mixed with streaks of blood. Necrotic pieces of cartilage are found not infrequently in the sputum.

But sometimes the spontaneous rupture of the abscess becomes dangerous because pus and pieces of cartilage are wedged between the true vocal cords and give rise to suffocation.

The development of internal or external fistulæ is also observed; this is not a favorable termination.

Wilkes and Ziemssen observed extensive emphysema of the skin as the result of laryngeal fistula.

Even if all these dangers have been passed, the rupture of the abscess usually exercises a very unfavorable influence on the general nutrition. Decomposition of the pus is apt to occur, followed by febrile movement and emaciation. The bronchi and lungs become affected by the downward flow of the pus, and death follows from pulmonary inflammation, abscess, gangrene, or phthisis.

When the termination is most favorable—from the formation of a cicatrix—stenosis occurs, with immobility of the cartilages and vocal cords, and the like.

IV. DIAGNOSIS.—The diagnosis is easy if we take into consideration the history and course of the disease, and the local changes. Laryngoscopic examination is the best diagnostic aid.

V. PROGNOSIS.—The prognosis is not favorable. In many cases it is bad on account of the primary disease, but it is usually gloomy even in primary perichondritis.

VI. TREATMENT.—So long as inflammatory symptoms are present, without evidences of abscess formation, local antiphlogistics may be employed. These include: ice-compress or ice-bag to the throat, pieces of ice or ice-water internally, leeches (three to four, repeated for several days), inunctions with tinct. iodine or ol. crotonis and turpentine in equal parts (3 drops). In syphilitic individuals, anti-syphilitic medication is indicated.

If an abscess has formed, an incision should be made with a curved knife, covered with adhesive plaster as far as the tip, and introduced with the aid of the laryngoscope. This should be followed by inhalation of disinfectants (acid. carbolic. 2%, acid. boric. 2-4%, natr. benzoic. 2-3%, alumina acetica, 3%, etc.). External abscesses must also be opened early.

If there is danger of suffocation, which cannot be relieved by opening the abscess, tracheotomy must be performed. The tracheal canula must not infrequently be worn a long time, sometimes during the patient's whole life. Its premature removal may render tracheotomy again necessary.

In desperate cases, the question of extirpation of the larynx may be taken into consideration.

#### 4. *Paralysis of the Laryngeal Muscles.*

I. ETIOLOGY.—Paralysis of the laryngeal muscles is not a rare affection. It is more frequent in adults than in children, and the male sex has an undoubted predisposition to certain forms of the disease.

The paralysis may be neuropathic or myopathic. In many cases the diagnosis can be made only after microscopical examination of the nerves and muscles.

In the neuropathic form, the disease may be located in the central nervous system or in the peripheral course of the nerves. An anatomical lesion is not always demonstrable.

The laryngeal muscles are supplied by the pneumogastric and spinal accessory nerves. The external branch of the superior laryngeal nerve supplies the crico-thyroid, perhaps also the depressor epiglottidis muscle; its internal branch sends sensory fibres to the mucous membrane of the upper laryngeal cavity as far as the free edge of the vocal cords. The recurrent laryngeal nerve supplies the larger number of laryngeal muscles, its motor fibres being derived from the spinal accessory.

The paralysis of the muscles is divided into three classes—respiratory, phonic, and mixed. The first class includes paralysis of the crico-arytenoidei postici which separate the vocal cords during inspiration. The other laryngeal muscles are devoted exclusively or mainly to phonation. In the mixed form of paralysis, both groups of muscles are affected.

The paralysis may be complete or incomplete, unilateral or bilateral, restricted to a single muscle or to certain groups of muscles.

The following are the causes of paralysis of the laryngeal muscles:

*a. Diseases of the central nervous system.*

Paralysis of the laryngeal muscles is observed most frequently in diseases of the medulla oblongata and pons (progressive muscular atrophy, bulbar paralysis, multiple cerebro-spinal sclerosis, tabes, etc.). It is also observed occasionally in diseases of the cerebrum (hemorrhage).

*b. Diseases of the vago-accessory trunk or its branches.*

The lesion is sometimes situated high up, even within the skull (tumors of the posterior fossa and foramen lacerum). Cases have also been observed in which the roots of the nerve trunk were thin, gray, and atrophic. Laryngeal paralyses have been known to develop after surgical operations or accidental injury to the neck (lesion of the trunk of the pneumogastric). Compression of the vagus and paralysis of all or a few laryngeal branches is very often the effect of tumors of the neck, oesophageal cancer, tumors of the thyroid gland, mediastinal tumors, aortic aneurism (compression of the left recurrent nerve), aneurisms of the innominate or right subclavian arteries, perhaps dilatation of the ductus arteriosus, pericarditis, pleurisy, pyo-pneumothorax, enlargement of the bronchial glands, pleuritic adhesions, etc. Heller described cancerous degeneration of the pneumogastric and recurrent nerves as causes of paralysis of the laryngeal muscles.

Rosenbach and Simon have called attention to the fact that in central paralyses or those caused by affections of the vago-accessory trunk, the crico-arytenoidei postici are the first muscles affected.

*c. General neuroses, particularly hysteria, sometimes epilepsy.*

*d. Reflex paralysis.*

Laryngeal paralysis has been observed in affections of the tonsils. Worms, uterine affections, etc., have also been mentioned as causes.

*e. Poisoning.*

The affection has been observed in lead, opium, belladonna, stramonium, and cannabis indica poisoning.

*f. Infectious diseases.*

The best known laryngeal paralyses are those following diphtheria, but they also occur after typhoid fever, relapsing fever, cholera, variola, pertussis, erysipelas, dysentery, syphilis, malaria.



### g. Diseases of the larynx itself.

Laryngeal catarrh, perichondritis, and operations on the larynx are not infrequent causes of paralysis. Ott described a case in which the ingestion of a large mouthful of food produced paralysis of the crico-arytænoidæi postici, probably by traumatism. Navratil observed destruction of the laryngeal muscles by trichina as the cause of paralysis. Loud and continued singing and shouting may give rise to paralysis. It has also been observed occasionally after tracheotomy.

h. Cold is said to produce rheumatic paralysis directly.

i. Mental excitement has also been mentioned as a cause.

k. Some writers describe congenital paralysis of the laryngeal muscles.

II. ANATOMICAL CHANGES.—In some cases, the muscles and nerves are said to have been unchanged. In others, degenerative and atrophic changes were found in the nerves alone. In still others, the muscles had undergone fatty degeneration, granular degeneration, nuclear proliferation, and connective tissue hyperplasia. These changes will develop secondarily if neuropathic paralysis has existed for some time.

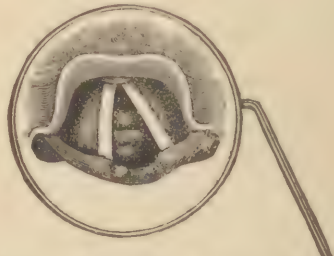
III. SYMPTOMS AND DIAGNOSIS. *a. Paralysis of the Crico-Arytænoidæi Postici Muscles.*—This form of paralysis is easily recognized. There is inspiratory laryngeal stridor (inspiratory dyspnoea) with intact or nearly intact phonation, while the laryngoscope shows that the vocal cords do not separate during inspiration, but, on the contrary, approach one another (Fig. 39). If the paralysis is unilateral, the vocal cord on the paralyzed side remains in the median line during inspiration, while the other one moves to the outer side (Fig. 40).

FIG. 39.



Laryngeal image in paralysis of both Mm. crico-arytænoidæi postici. Position during inspiration.

FIG. 40.



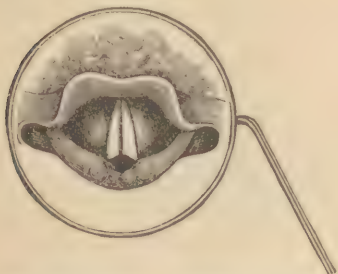
Laryngeal image in paralysis of the right crico-arytænoidæus posticus. Position during inspiration.

The clinical phenomena are readily explained by the function of the muscles. When they are paralyzed, the vocal cords are not separated during inspiration in order to allow the entrance of air into the deeper air passages, but they remain close together; indeed, they may be pressed against one another by the inspiratory current of air. If inspiration is not performed slowly and cautiously, the free edges of the vocal cords are brought into such close apposition by aspiration that the patient is in danger of suffocation, particularly when considerable catarrhal swelling of the vocal cords is also present. Expiration is undisturbed. Phonation is unaffected or very little impaired, the latter particularly in the production of high notes, since the arytenoid cartilages are rendered somewhat more mobile on account of the paralysis.

*b. Paralysis of the arytenoid muscles* is shown by the fact that, on

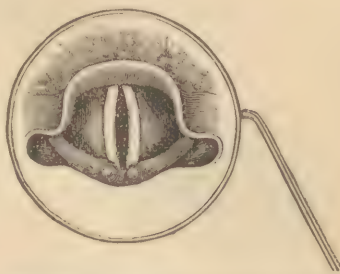
laryngoscopic examination, the posterior third of the rima glottidis remains open during phonation in the shape of a triangular slit (Fig. 41).

FIG. 41.



Laryngeal image in paralysis of the arytenoid muscles. Position during phonation.

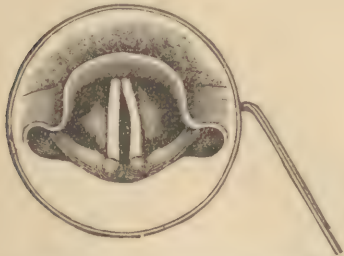
FIG. 42.



Laryngeal image in paralysis of both thyreo-arytenoid interni. Position during phonation.

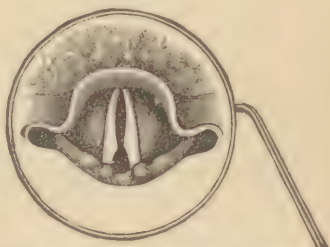
The open portion is known as the cartilaginous glottis, also glottis respiratoria, *i. e.*, that portion between the edges of the arytenoid cartilages. Its closure is effected by the apposition of the inner edges of these cartilages through the contraction of the arytenoid muscles. The paralysis is often the result of inflammatory irritation. Other characteristic symptoms are absent.

FIG. 43.



Laryngeal image in paralysis of the left internal thyreo-arytenoid muscle. Position during phonation.

FIG. 44.



Paralysis of both internal thyreo-arytenoid and both arytenoid muscles. Position during phonation.

*c. Paralysis of the Internal Thyro-Arytenoid Muscles.*—With the laryngoscope it is found that the anterior two-thirds of the rima glottidis are not situated in close apposition, but that there is an oval slit between them (Fig. 42). When the paralysis is unilateral, the free edge of the paralyzed vocal cord is a little bent, the healthy one is straight (Fig. 43). The voice is hoarse, partly as the result of catarrh.

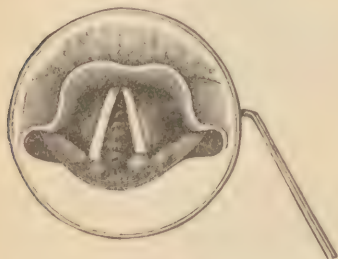
The arytenoid and internal thyro-arytenoid muscles are often paralyzed together, especially as the result of catarrh. The entire glottis then remains open during phonation, its two parts separated from one another by a slight projection corresponding to the processus vocalis of the arytenoid cartilages.

*d. Paralysis of the lateral thyro-arytenoid muscles.* These muscles are closers of the vocal cords, but their isolated paralysis cannot be recognized with the laryngoscope. This is also true of

*e.* Paralysis of the external thyro-arytaenoid muscles.

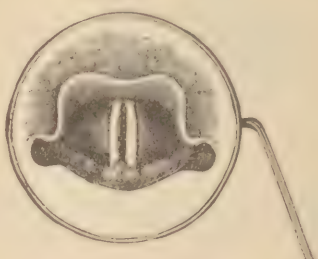
*f.* Paralysis of the entire recurrent laryngeal nerve. The paralyzed vocal cord is incapable of movement. During respiration and phonation it remains immovable in a situation midway between the median line and inspiratory external position (Fig. 45) (cadaver position). Respiration is unimpeded. During phonation the healthy vocal cord passes be-

FIG. 45.



Laryngeal image in paralysis of the left recurrent nerve. Position during inspiration.

FIG. 46.

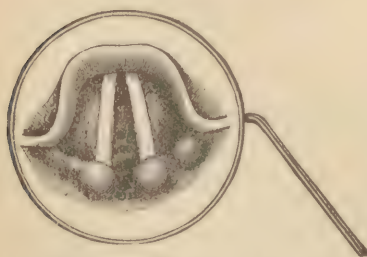


Laryngeal image in paralysis of the left recurrent nerve. Position during phonation with crossing of the arytaenoid cartilages.

yond the median line and comes in contact with the paralyzed one, thus rendering phonation and cough possible. The voice is generally hoarse, as the paralyzed vocal cord is incapable of regular vibrations. Furthermore, the arytaenoid cartilages cross one another during phonation, the one on the healthy side being generally in front of the other (Fig. 46).

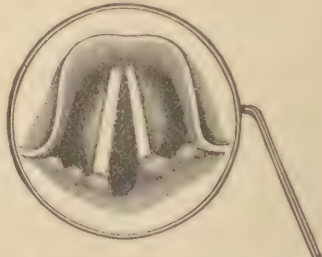
In bilateral paralysis of this nerve, both vocal cords remain in the cadaver position during phonation and respiration (Fig. 47). There is no dyspnoea, on account of the wide separation of the glottis, but during forced inspiration creaking noises may be produced from the vibration of the free edges of the vocal cords, which have not been put on the stretch. The patients are incapable of speaking aloud or of coughing. Their speech resembles an inarticulate grunting; whispering can be done better. All attempts at speaking strain the patient exceedingly because so much air is wasted on account of the patency of the glottis.

FIG. 47.



Laryngeal image in bilateral paralysis of the recurrent nerve. Vocal cords in cadaver position.

FIG. 48.



Laryngeal image in paralysis of the left recurrent nerve with atrophy of the paralyzed vocal cord. Position during inspiration. After Ziemssen.

*g.* Paralysis of the crico-thyroid muscles. These muscles are tensors of the vocal cords and act in the production of high notes. This be-



comes impossible or is rendered very difficult, according as the muscles are paralyzed or paretic. In addition, it will be found that when the tip of the index finger is inserted in the space between the thyroid and cricoid cartilages, the former does not approach the latter in the attempt to produce high notes.

*h.* Paralysis of the thyro-ary-epiglottic muscles. The function of these muscles, to which Merkel applied the term depressor epiglottidis, is to draw the epiglottis backward over the entrance to the larynx during deglutition, and to prevent the entrance of food into the laryngeal space. When the muscles are paralyzed, a part of the food enters the larynx, the patients swallow the wrong way, and suffer from spasmodic cough and dyspnoea, or pneumonia and gangrene of the lungs develop at a later period. With the laryngoscope the epiglottis is found to be motionless.

*i.* Paralysis of the entire superior laryngeal nerve. The symptoms include those mentioned under *g* and *h*. In addition, there is anaesthesia of the laryngeal mucous membrane as far as the vocal cords and extending upwards into the lower pharyngeal region. Paralysis of this nerve is most frequent in diphtheria. In some cases the crico-thyroid and thyro-ary-epiglottic muscles seem to be supplied by the recurrent nerve, because the symptoms of paralysis of these muscles have been observed in paralysis of the recurrent nerve.

Cases of intermittent paralysis of the vocal cords have been described. The paralysis begins at a certain time of the day and lasts for a variable period, after which it disappears. No positive relation to malarial infection has been demonstrated. It should not be mistaken for hysterical paralysis, the intermissions in which are entirely irregular.

Among the sequelae should be mentioned atrophy of the vocal cords as the result of long standing paralysis (Fig. 48).

IV. PROGNOSIS.—This depends, in the main, on the primary affection. The prognosis also varies according to the function of the paralyzed muscles. The most dangerous form is paralysis of the crico-ary-tenoidei postici, which may give rise to danger of suffocation at any moment. Paralysis of the depressor glottidis not alone produces great distress, but may also give rise to secondary pneumonia or gangrene of the lungs.

V. TREATMENT.—Prophylactic measures must be adopted by those patients suffering from laryngeal diseases, in whom a fresh cold or over-exertion of the voice is apt to be complicated with paralysis of the muscles.

The general treatment depends on the primary affection, but it will be impossible for us to enumerate here all the possible external and internal remedies. In diphtheritic paralysis, subcutaneous injections of strychnine have been successfully employed. In intermittent paralysis, which did not yield to quinine, Levison effected a cure by the administration of arsenic (aq. amygdal. amar., liq. kali arsenicos., ãã 5.0. M. D. S. Ten drops t. i. d. after meals).

The following methods must be considered in the local treatment.

*a.* Brushing with nitrate of silver or insufflation with alum (0.1), tannin (0.1), acetate of lead (0.05), iodoform (0.05), etc. In part such remedies relieve the catarrh, in part they irritate the vocal cords and thus stimulate the paralyzed muscles to action. With the same end in view, Rossbach applies a sound to the vocal cords, and administers emetics.

Insufflation may be performed with Rauchfuss' powder blower, composed of a curved hard-rubber tube carrying a bulb at one end (Fig. 49). Near the latter the tube presents an oval opening, which can be closed by a sliding tube. The powder used in insufflation is poured into the opening and the sliding tube drawn over it. While the patient opens the mouth and protrudes the tongue, the instrument is introduced into the mouth until the free end projects over the entrance to the larynx. The patient is then directed to take a full breath and, at the same time, the bulb is compressed. At first this may cause spasm of the laryngeal muscles and fear of suffocation, but these symptoms soon subside. The insufflation may be repeated daily.

FIG. 49.



Rauchfuss' powder blower.

#### 6. Gymnastics of the larynx (v. Bruns).

A laryngoscopic examination is made, and the patient at the same time directed to utter the vowels aloud.

#### c. Compression of the larynx (Olliver and Gerhardt).

The cornua of the thyroid cartilage are compressed between the thumb and index finger, the patient being directed to inspire deeply, and to utter the vowels aloud.

d. Compressed air has been employed successfully in a few cases.

#### e. Electricity.

FIG. 50.



Laryngeal electrode ( $\frac{1}{2}$  natural size). The electrode is introduced into the larynx, while the finger is pressed upon the spring in order to keep the circuit open. The circuit is closed when the electrode is in position.

The faradic current should be used, one pole being placed on the integument of the neck or within the larynx. Intra-laryngeal faradization requires a special electrode (Fig. 50). When applied externally, the current should be strong enough to produce contraction of the thumb muscles; when applied within the larynx, to produce contraction of the frontal muscle. To stimulate the superior laryngeal nerve, the electrodes should be placed on the superior cornua of the thyroid cartilage; to stimulate the recurrent nerve, they are placed on the inferior cornua.

The inferior laryngeal nerve is reached inside of the larynx at the sinus pyriformis. Stimulation of individual muscles is effected at the following points: *a.* Crico-arytænoidei pestici—posterior plate of the cricoid cartilage immediately behind the arytenoid cartilages. *b.* Arytænoids—middle of the inter-arytenoid region. *c.* Internal thyro-arytænoids—free edge of the vocal cord. *d.* Thyro-ary-epiglottici—free edge of the corresponding ligament. The indifferent electrode is placed high up on the back of the neck.

In hysterical paralysis, M. Meyer employed the electrical moxa, *i. e.*, a wire brush electrode applied so close to the skin as to permit the passage of sparks.

Long-standing laryngeal paralyses are sometimes cured by strong mental excitement.

If suffocation threatens in paralysis of the crico-arytænoidei postici, tracheotomy must be performed. Indeed, the dyspnœa may appear so rapidly and unexpectedly that the advisability of performing the operation at the onset should be taken into consideration. In one case, Sidlo successfully passed a catheter through the rima glottidis, and thus secured a supply of air to the lungs. In paralysis of the thyro-ary-epiglottic muscles the patient should be nourished with the aid of the œsophageal sound. This procedure may become dangerous if the laryngeal mucous membrane is anæsthetic, because the sound is then apt to enter the larynx.

### 5. *Spasm of the Glottis.*

(*Laryngismus stridulus s. Asthma laryngeum.*)

I. ETIOLOGY.—This disease occurs almost exclusively during childhood. It consists of brief attacks of spasm of the constrictors of the glottis and the diaphragm, *i. e.*, in the distribution of the recurrent laryngeal nerve. The disease is a functional neurosis.

It is most frequent from the age of six to twenty-four months; it is rare prior to dentition or after the second year.

It is more frequent in boys than in girls, especially in rachitic or scrofulous children (probably nine-tenths of all cases occur in rachitic children).

Some authors attribute the disease to hereditary influences. It is true that several children in the same family often suffer from spasm of the glottis, but this is the result of rachitis, either inherited or acquired as the effect of improper nutrition and mode of life.

The disease often develops in extremely obese but pale children. In addition, they often present evidences of nervousness (restless sleep, capriciousness, excitability). The individual attacks often appear to develop spontaneously. In some cases a cold seems to act as an etiological factor. The disease is observed most frequently in the cold months, and occasionally is almost epidemic. Indigestion (vomiting, constipation, diarrhœa) may also give rise to an attack of spasm of the glottis. Dentition also exerts an influence in this direction. An attack may also be the result of mental excitement (fright, joy, crying), nursing at the breast, depression of the tongue with a spatula, tossing the infant in the arms, etc. Joffroy states that he has seen attacks in tracheotomized children after removal of the tracheal canula (?), but it seems certain that the attack is produced by catarrh of the laryngeal mucous membrane or loud crying.

Fleisch describes one variety (spasmus glottidis ablactatorum) which occurs in children of well-to-do parents upon attempting to wean the child from the breast.

II. SYMPTOMS.—Attacks of arrested respiration constitute the chief symptom of spasm of the glottis. They occur suddenly in the midst of health or during sleep. Children in arms suddenly breathe irregularly and pantingly, the individual respirations become deeper and deeper, and



soon cease. The little patients close their eyes or roll them around; the head sinks on the shoulder of the nurse, they grow pale and livid, and lie as if dead. The action of the heart becomes excited and often irregular, the pulse becomes very frequent and remarkably small. The cervical veins enlarge. Percussion shows that the upper border of the liver, *i. e.*, the diaphragm, is very low during the attack. Sometimes there is an involuntary evacuation of urine and feces. In a little while, as a rule, the respirations again begin, with a slow, deep inspiration, accompanied by whistling or crowing stridor, which may be heard through a number of rooms. The child comes to, opens his eyes, and soon is as cheerful as before the attack.

The attack generally lasts only a few seconds, but sometimes half a minute. It may be so brief that its recognition requires some attention. A single attack may alone occur, or a large number are observed in the course of a single day, or they are repeated at intervals of a few weeks or months.

The spasm sometimes extends to the muscles of the limbs or trunk, giving rise to convulsions, which may occur before, during, or after the suspension of respiration. The spasmodic movements are observed most frequently in the hands and feet. The thumb is bent into the hollow of the hand, the other fingers are separated and extended. In the foot, we notice plantar extension of the great toe, *séparation* and dorsal flexion of the other toes, and dorsal flexion of the entire foot. Unconsciousness and general convulsions (*eclampsia infantum*) are occasionally observed. Death may occur during an attack from suffocation or *eclampsia*, or a condition of *marasmus* to which the patient succumbs may develop.

In a few cases, Cohen and Ebstein observed spasm of the thyro-ary-epiglottic muscles, by which the epiglottis was drawn backwards, thus increasing the danger of suffocation.

III. ANATOMICAL CHANGES.—The post-mortem appearances hitherto observed have failed to explain the conditions present during life. The following lesions have been observed, but not constantly:

Rachitic changes in the bones, hydrocephalus, cerebral congestion, meningeal hemorrhage, meningitis, enlargement of the thymus gland, enlargement of the thyroid gland, enlarged bronchial and tracheal lymphatic glands, patency of fetal channels of circulation, fatty liver, enlargement of the lymph follicles of the intestine and of the mesenteric lymphatic glands.

All the theories which have been advanced in explanation of the disease are insufficient. We have to deal with a neurosis which is sometimes central, sometimes peripheral, sometimes reflex in its origin.

IV. DIAGNOSIS.—The disease is easily recognized, as the brief attacks of arrested respiration, with the crowing or whistling inspiration at the return of respiration, are so characteristic that differential diagnosis need not be considered.

V. PROGNOSIS.—The prognosis must be given with caution. Some authors report a mortality of ninety per cent, others a mortality of eight per cent, or even less. As a rule, the prognosis is so much more unfavorable the younger the child, the longer the attacks, and the greater the tendency to *eclampsia*. It is also said to be more favorable in males than in females.

VI. TREATMENT.—When other members of the family have suffered from spasm of the glottis, prophylactic measures should be adopted.

The dietetic and psychical training should be carefully regulated, cold and indigestion avoided, and rachitis or scrofula treated in the usual manner.

During the attack, the child should be raised up, and, later, he should be placed on the side, not on the back. The room should be large, the window opened, the chest sprinkled with cold water, the soles of the feet or nasal mucous membrane tickled, or the child should be put in a warm bath, and douched with cold water. The inhalation of ammonia or acetic acid often produces reflex acts of respiration. If the attack is prolonged, the finger should be introduced into the throat, in order to raise the epiglottis if it has been forced downward.

To prevent the return of the attack, we must inquire carefully into the causes. If the result of indigestion, an enema should be given; if the result of constipation, order a laxative (calomel, 0.1) or, perhaps, an emetic.

If the children are very nervous, we may administer for a long time nervines and mild narcotics (for example, sol. kali. bromat., 5.0 : 100. D. S., one teaspoonful every two hours).

Asafœtida, musk, zinc, silver, copper, atropine, valerian, cannabis indica, digitalis, quinine, are also recommended.

In spasmus glottidis ab lactatorum, the child should again be nursed at the breast. Anæmic, scrofulous, and rachitic conditions require appropriate treatment.

If the attacks recur repeatedly in the course of a single day, we must resort to the use of chloroform, ether, or chloral hydrate (chloroform and ether inhaled until beginning narcosis, and chloral hydrate 0.5 per enema). In many cases, tracheotomy cannot be performed on account of the age of the patient: at all events, it should always be combined with faradization of the phrenics. This requires a strong current and large electrodes, which are placed at the outer edges of the sterno-mastoids, above the omo-hyoid muscles. The current should be opened at regular intervals of one to two seconds. Expiration is aided by vigorous compression of the bowels.

### 6. *Spasm of the Laryngeal Muscles.*

1. Unlike spasm of the glottis, spasm of the laryngeal muscles is more frequent in adults, particularly in hysterical patients. It is rarer in epilepsy, and is also observed in hydrophobia, lead poisoning, pressure on the vagus or recurrent laryngeal nerve by tumors or goitre, after inspiration of irritating gases or the entrance of foreign bodies. In one case, the spasm occurred whenever the patient had a spoiled stomach and sour eructations, during which the gases often passed into the larynx. Diseases of the larynx itself, for example œdema of the glottis or polypi, may act in the same way.

The closers of the glottis are usually affected, giving rise to inspiratory dyspnoea. The symptoms may be very similar to those of paralysis of the crico-arytænoidei postici, but they continue only for a short time. The spasm should be treated by narcotics (chloroform, chloral), the action of which may be aided by the application of galvanism. This condition may lead to death from suffocation, as L. Meyer observed in hysterical cases.

2. Schnitzler applied the term phonic spasm of the glottis, aphonia spastica, to a condition of disturbed co-ordination of the muscles of the

vocal cords. In attempts at phonation the tensors of the vocal cords contract spasmodically, bringing them into close apposition and rendering loud phonation impossible. The patients can only whisper. The explosives—p, b, d, t, k, g—are best enunciated. The patients are often hysterical and nervous, and complain of a feeling of constriction in the larynx and pressure on the chest. The disease may also result from excessive use of the voice. Mackenzie observed it with relative frequency among clergymen; also in two individuals, whose families were deaf. Cold has also been mentioned as a cause. Rest to the voice, electricity, and treatment of the primary affection are indicated.

3. The abnormally high-pitched voice of many adults is attributed by Pieniazek to excessive action of the tensors of the vocal cords. This also occurs in women who speak or shout a great deal.

### 7. *Sensory Disturbances of the Laryngeal Mucous Membrane.*

1. Anæsthesia of the laryngeal mucous membrane has been observed in diphtheria, hysteria, and bulbar paralysis. In diphtheria the pharynx, as a rule, is also anæsthetic. When the anæsthesia is confined to the superior laryngeal nerve, the mucous membrane is anæsthetic only as far as the free edges of the vocal cords. Sometimes the tracheal mucous membrane is also affected.

As a rule, the motor fibres are also paralyzed, and the epiglottis is found to be immovable. Food may therefore enter the larynx and, as the mucous membrane is anæsthetic, enter the lower air passages. Ott recently described unilateral anæsthesia of the larynx from syphilitic degeneration of the right pneumogastric. The treatment consists of faradization of the mucous membrane, hypodermic injections of strychnine, the administration of iron, and feeding with the œsophageal sound.

In one case Schnitzler observed anæsthesia associated with neuralgic symptoms (anæsthesia dolorosa).

2. Hyperæsthesia and paræsthesia of the laryngeal mucous membrane are frequent in catarrh or ulceration of the larynx. In hysterical individuals they may appear as an independent disease. The patients may be so annoyed by tickling, burning, or the sensation of a foreign body in the larynx, that they speak in a whisper for weeks or months (phonophobia), in order to be relieved of their supposed serious disease. In a few cases E. Fraenkel observed painful points in the neck. The voice is clear, and the laryngoscopic appearances negative. We may order inhalations of potassium bromide (5.0 : 100), morphine (0.02–0.1 : 100), pencil the larynx with bromide of potassium (5.0; glycerini 25.0) and exercise the patient daily in loud speaking. Lewin and Waldenburg saw good effects from conium maculatum (ext. conii 0.25–0.5 : 100 for inhalation). Schnitzler recommends the application of morphine or chloroform. Solutions of nitrate of silver have also been applied. I have seen good effects from inhalations of tannin (4.0 : 150).

The hyperæsthesia is occasionally intensified into well-marked neuralgia.

### 8. *Laryngeal Cough.*

This is an independent neurosis, not associated with changes in the air passages. The patients (usually females between the ages of fifteen and twenty-five years) are pale and nervous, and suffer not infrequently



from other hysterical symptoms. The disease is attributed by some patients to cold, drinking cold beverages, and straining the voice.

In some cases paroxysms of coughing occur, not infrequently during sleep. Other patients cough almost uninterruptedly. Sometimes the cough is barking, whistling, stertorous. Expectoration is very slight or entirely absent. The affection may last for months.

Change of residence usually affords relief (Mackenzie recommends sea voyages). I have seen no good effects from narcotics. Lasegue reports a cure from the use of belladonna.

### PART III.

#### DISEASES OF THE TRACHEA.

Diseases of the trachea are rarely independent. As a rule, they are propagated either from the larynx or the bronchi. The secondary implication of the trachea is then either entirely latent or of subordinate importance. We refer the reader, accordingly, to the sections on laryngeal and bronchial diseases.

Herterich recently described a case of mycosis tracheæ. The nose, mouth, pharynx, and larynx were intact, but the trachea presented excoriations, the patient coughed, and the sputum contained gray, hard masses. Under the microscope these were found to contain numbers of eurotium aspergillus. Recovery followed inhalations of iodine vapor, t. i. d. for a few minutes.

Hindelang described a case in which the fungi did not develop in the trachea, but were inhaled and deposited in part in the trachea. Recovery after inhalation of carbolic acid and insufflation of iodoform.

### PART IV.

#### DISEASES OF THE BRONCHI.

##### *Bronchial Catarrh.*

##### *(Bronchitis catarrhalis.)*

**I. ETIOLOGY.**—Bronchial catarrh is one of the most frequent diseases. It may be either acute or chronic, the former variety lasting for a few days or weeks, the latter for many years, even for life. It may be bilateral or unilateral, affect only a few branches, or the large bronchi alone, or only the finer bronchi (bronchioles). Catarrh of the large bronchi is associated usually with a similar affection of the trachea (tracheo-bronchitis). Catarrh of the finer bronchi is especially dangerous, because an accumulation of secretion, or marked swelling of the mucous membrane, readily leads to complete occlusion of the bronchial lumen. This form is also known as bronchiolitis, s. bronchitis capillaris.

From an etiological standpoint, bronchial catarrh may be primary or secondary.

A cold is the most frequent cause of primary bronchial catarrh. A wetting, sudden cooling of the heated skin, careless bathing in cold water, staying in damp, draughty rooms, incautious change of clothing, may give rise to bronchial catarrh.

The seasons and winds exert an undeniable effect. Bronchial catarrh is especially frequent in autumn and spring, and during the prevalence of north and northeast winds. In some parts of the tropics, bronchitis is a rare disease. It increases in frequency towards the colder regions.

A. Hirsch has shown that the frequency of bronchial catarrh depends directly on the amount of moisture in the air. The dryer the air, and the greater the difference between the dew-point and the ordinary temperature of the air, the more unfavorable are the conditions for the development of the disease.

In many cases, there is a predisposition of the respiratory mucous membrane to react with bronchial catarrh to certain injurious influences. There are a number of predisposing factors, prominent among them being age. Children and old people are specially predisposed. Bronchial catarrh is very rare in the first six months of life, probably because the children are carefully protected. From that period until the end of the third year, the disease is very frequent. It then grows more infrequent. Many children also suffer from this affection at the period of dentition.

The varying predisposition at different periods of life depends on the greater or less power of resistance of the organism, particularly of the bronchial mucous membrane. This also explains the effect of the constitution on the development of bronchial catarrh. Feeble, anæmic, chlorotic individuals manifest a marked predisposition to the disease. It is also observed very frequently in scrofula and rachitis. Long-continued and exhausting diseases and marastic conditions (cancer, syphilis, malaria, Bright's disease, diabetes, scurvy, gout, alcoholism, etc.) also furnish a marked predisposition. The slight power of resistance of the organism is sometimes the result of defective bodily training, of an enfeebling, enervating mode of life.

Geigel showed that in Würzburg legitimate children suffered more than illegitimate children from bronchial catarrh during the first year of life. The reverse held good with regard to digestive disorders. This has been explained on the ground that the legitimate children are more carefully looked after, and are thus apt to be enfeebled. The children are very often protected anxiously from every breath of air, and at the same time are allowed to run about with bare legs.

Primary bronchial catarrh also includes those cases which are the result of impurities in the air. This may follow mechanical irritation of the bronchial mucous membrane by substances in the shape of dust, or the inhalation of irritating gases.

Heit proved that the inhalation of vegetable dust is the most dangerous; next come metallic and animal dust; finally, mineral dust. Certain occupations which lead to the inhalation of a dust-laden atmosphere have long been known to produce the disease. This category includes millers, stone-cutters, coal-miners, bakers, brush-makers, spinners, weavers, metal-workers, cigar-makers, etc. Among injurious gases may be mentioned chlorine, acetic acid, hydrochloric acid, sulphuric, sulphurous, and nitrous acids.

Toxic broncho-catarrh is a third form of primary bronchial catarrh. It is produced by the internal administration of certain drugs.

Iodide of potassium produces bronchial and conjunctival catarrh in certain individuals even when given in small doses. Stille has recently shown that bromide of potassium may also give rise to obstinate bronchial catarrh. This possesses great importance, because the bromide may diminish the sensibility of the respiratory mucous membrane to such an

extent that an excessive amount of secretion may accumulate in the bronchi, and give rise to danger of suffocation.

Those catarrhs which follow the entrance of foreign bodies are purely mechanical in their nature.

Finally, primary bronchial catarrh may be the result of straining in singing, speaking, or playing on wind-instruments.

This is observed most frequently in clergymen, teachers, actors, and musicians. As a rule, catarrh of the larynx first develops and then extends to the bronchial mucous membrane, but in other cases the bronchial catarrh may develop primarily. Under such circumstances, the respired air, which remains beneath the vocal cords for an unusually long time and often under abnormally high pressure, acts as a mechanical irritant on the bronchial mucous membrane.

Secondary bronchial catarrh is observed most frequently as the result of pulmonary diseases.

Both acute and chronic diseases of the lungs are associated generally with bronchial catarrh. In acute diseases, the inflammation is usually propagated from the alveoli to the bronchial mucous membrane; in the chronic diseases, the bronchial catarrh is generally the result of circulatory disturbances. Only a portion of the blood in the bronchial veins flows into the vena cava through the vena azygos and vena hemiazygos. Another part flows into the pulmonary vessels and then into the heart. Furthermore, branches of the bronchial arteries communicate in the interstitial tissue of the lungs with small branches of the pulmonary artery. Hence changes in the pulmonary parenchyma affect the distribution of blood to the bronchial mucous membrane, and may give rise to catarrhal changes.

Secondary stasis catarrh is observed not infrequently in diseases of the heart.

Since a portion of the bronchial venous blood flows into the left, another part into the right auricle, there is evidently a favorable opportunity for stasis of blood and bronchial catarrh, because diseases of the right as well as the left heart must exercise an injurious influence. The most violent and obstinate bronchial catarrhs are observed in mitral lesions, but they also occur in other cardiac diseases as soon as the flow of blood from the pulmonary veins or venæ cavæ is impeded.

Diseases of the abdominal organs (tumors, ascites, etc.) occasionally give rise to the disease. We believe that the impaired motion of the diaphragm, and consequently the slight expansion of the lungs, diminished aspiration and propulsion of the blood in the pulmonary artery, give rise to stasis catarrh, because the impeded flow of blood from the pulmonary artery causes stasis in the venæ cavæ and bronchioles.

Bronchial catarrh is sometimes propagated from inflammatory conditions of the larynx and trachea.

Finally, secondary bronchial catarrh is observed in many febrile infectious diseases: measles, scarlatina, small-pox, whooping-cough, influenza, malaria, typhoid fever, relapsing fever, etc. In these cases it is probable that low organisms have a direct injurious effect on the bronchial mucous membrane.

II. ANATOMICAL CHANGES.—In acute catarrh of the large bronchi, the mucous membrane is very red, the vessels of the mucosa and submucosa are unusually large, and in many places are visible to the naked eye. Other places present a diffuse redness. The hyperæmia is found



sometimes in patches, sometimes diffused uniformly over the mucous membrane. Subepithelial ecchymoses may be seen in a few places.

The inflamed mucous membrane is also unusually swollen and succulent. The tissue appears loosened, extremely juicy, and also unusually brittle, and but little resistant. The surface often has a dull, velvety gloss, and, in the beginning, may be very dry.

After the catarrh has lasted for some time, as a rule, the secretion of the mucous membrane is increased. Upon the inner surface of the bronchi is found a glassy and tough or a greenish, opaque, more purulent fluid. The mouths of the glands of the mucous membrane are filled occasionally with fine, pearly drops of mucus.

These lesions often disappear, in part, after death. This is particularly true of the swelling of the mucous membrane, but the congestion may also be unexpectedly slight.

Under the microscope the vessels of the mucosa and submucosa are found unusually full, and they also give evidences of the emigration of white blood-globules. The cells of the subepithelial layers of the mucous membrane are in a condition of proliferation, and there is marked increase of the nuclei of the epithelium cells. In severe catarrh, there may be an accumulation of round cells in the peribronchial connective tissue.

In acute catarrh of the finer bronchi, it is often found, on opening the thorax, that the lungs, instead of collapsing, protrude still further from the chest. This phenomenon must be explained by the fact that the finest bronchial tubes are occluded, so that the air cannot escape from the alveoli. In addition, the air must be inclosed in the alveoli under unusually high pressure. In capillary bronchitis there are often other changes in the lungs. Some portions are often very much distended and emphysematous, particularly the anterior median and inferior borders of the lung. Other parts of the lung appear empty of air, collapsed, and very congested. These parts can be filled with air on blowing into a main bronchus. Such changes are most frequent in the posterior and inferior portions of the lungs, and are the result of complete absorption of air from alveoli which no longer communicate with the bronchi. Not infrequently we find hard, dark-red non-aerated spots, which cannot be filled with air. These are foci of catarrhal pneumonia (broncho-pneumonia), which have developed either from the propagation of the inflammation from the terminations of the bronchi into the pulmonary alveoli, or from the aspiration of the infectious contents of the finer bronchi into the alveoli.

The pulmonary pleura, in spots, is occasionally ecchymotic and opaque, generally over atelectatic and broncho-pneumonic foci. According to Bahl's investigations, the bronchial arteries are intimately connected with the vessels of the pleura.

Pressure upon the cut surface of the lungs expresses from the finer bronchi a frothy, clear, glassy or pus-like fluid, which is also found to fill, to a greater or less extent, the lumina of the bronchi. The mucous membrane is also swollen and reddened. The secretion is sometimes collected into puriform plugs, which may be squeezed out of the lumen of the bronchi.

Traube showed that, in catarrh of the large bronchi, the secretion occasionally flows into the finer ones, and simulates bronchiolitis both during life and in the dead body. But in such cases the mucous membrane is not reddened or swollen.

Chronic bronchial catarrh is confined generally to the large and medium-sized bronchi. The mucous membrane, as a rule, has a grayish-red or brownish-red color. It is sometimes very thick, often from inflammatory hyperplastic processes. Papillary excrescences are sometimes formed. The mucous membrane not infrequently appears fenestrated by reason of longitudinal and transverse prominences, which arise in consequence of hyperplasia and increased prominence of the longitudinal and transverse bundles of elastic fibres, and of the atrophy of other parts, particularly the muscular layers, in consequence of the excessive expiratory pressure during coughing.

The mucous membrane is sometimes extremely pale and thin, and almost resembles a serous membrane.

This is observed most frequently in serous bronchorrhœa. As a rule, there is an abnormally profuse accumulation of secretion. This may be mucous, serous, muco-purulent, or almost entirely purulent. It may also undergo purulent decomposition.

In long-standing catarrhs, the mucous membrane may suffer superficial losses of substance. This occurs more rarely in acute bronchial catarrh. Ferrand has described several cases in old people, who presented striking cachexia and purulent secretion during life. The autopsy disclosed ulcerations of the mucous membrane near the bifurcation.

Bronchial dilatation (bronchiectasis) may develop in bronchial catarrh of long standing. The inflammatory hyperplasia may also extend to the peribronchial connective tissue, and lead to increase of the interstitial pulmonary tissue. Emphysema is also found very often as a sequel of chronic bronchitis.

The bronchial lymphatic glands often pass into an inflammatory condition through the medium of the bronchial lymphatics. In acute bronchitis, the glands are swollen, reddened, and succulent. In frequently relapsing or chronic catarrhs, the glands undergo hyperplastic changes, they become enlarged, pigmented, even cheesy and calcified.

III. SYMPTOMS. — Acute catarrh of the large bronchi may exist without any objective changes. The patients complain of an intolerable sensation of tickling, and a desire to cough, and often experience a sensation of rawness in the chest, along the upper part or the whole of the sternum. The integument over the sternum is sensitive to pressure. The patients are tormented with a cough, but are unable to expectorate. In many cases, the bronchitis is associated with tracheitis (tracheo-bronchitis). Under such circumstances, tracheoscopic examination may show the presence of a tracheitis. The mucous membrane of the trachea is seen to be very red, thick, and moist. Scattered accumulations of secretion are sometimes recognized in the shape of yellowish deposits and patches.

Febrile movement may be present, and is sometimes preceded by chilly sensations, or even a decided chill.

Catarrh of the large bronchi is recognized by auscultation only when rhonchi are heard. These may be dry or moist, according as the catarrhal secretion is very tough or fluid.

Tough bronchial secretion in the large upper air passages furnishes the conditions for the development of sonorous râles, while sibilant râles will be heard in catarrh of the deeper and smaller bronchi. Both kinds of râles are very often heard at the same time, or one form is heard after the other has lasted for some time.

The rhonchi are murmurs of stenosis. The deposit of tough secre-

tion on the bronchial mucous membrane, and the swelling of the latter, suddenly narrows the lumen of the tube, so that a whirl of air, and therefore a murmur, is produced below the stenosis during inspiration, and above it during expiration. A peculiar vibration, known as bronchial fremitus, is often felt on palpation.

When the bronchial secretion is fluid and loose, moist râles are heard. These may be coarse or moderately fine, or mixed; and according as they develop near the surface of the lung, or deeper in, they are clear or dull. But the râles of a simple bronchitis never acquire consonance or a metallic quality.

It is generally assumed that these râles are produced by the formation and bursting of vesicles in the loose fluid. Traube and Hertel showed that the acoustic impression of moist râles will also be produced if bronchial secretion is torn from one part of the mucous membrane by the current of air and carried to another place.

The secretion of acute bronchial catarrh is at first very tough, and later assumes a more fluid consistence. We find, therefore, that the disease usually begins with sonorous and sibilant rhonchi, and ends with moist râles. As a matter of course, dry and moist râles may be heard at the same time.

The normal respiratory murmur may also be changed. The expiratory murmur is very often found to be remarkably prolonged, evidently because the masses of secretion present great obstacles to the expiratory current of air. The inspiratory vesicular murmur also assumes a coarse or puerile character, partly on account of the unusually vigorous respiratory movements, partly because coarse murmurs are added to the vesicular breathing as a result of the bronchial stenoses produced by the deposit of secretion. Bronchial breathing is never heard in uncomplicated bronchitis.

If fever is present, the respiratory movements are more or less accelerated. Objective signs of dyspnoea are almost always absent. The prolongation of expiration as a result of bronchitis is usually recognized by the eye, and also by the hand when placed on the thorax. Percussion furnishes negative results.

Among the subjective symptoms the patients are annoyed chiefly by the cough. The desire to cough is so much more violent the more acute the catarrh, and the more the first portions of the bronchi are affected. In the beginning of bronchial catarrh the cough is much more violent than later, on account of the character of the sputum. At first nothing is expectorated, or the patients, with great difficulty, expectorate a colorless, transparent fluid, which is frothy on the surface and extremely tough. Upon the addition of acetic acid to the sputum, opaque, veil-like flocculi of mucus are formed. It consists in the main of mucin, and under the microscope is found to be extremely poor in round cells (Fig. 51).

Pavement epithelium cells from the buccal cavity are often found as accidental constituents. Ciliated epithelium cells from the bronchial mucous membrane are either absent, or present in scanty numbers. The ciliated edge is easily recognized after staining with methyl aniline. The cilia are sometimes lost, and the cells are then swollen and shaped like ordinary cylindrical epithelium.

If acetic acid is added to the microscopic preparation, web-like, filmy opacities, the result of coagulation of the mucin, are formed under the eye of the observer. The round cells swell, become transparent and



FIG. 51.



Mucous sputum in acute catarrh of the large bronchi. Magnified 375 times.

FIG. 52.



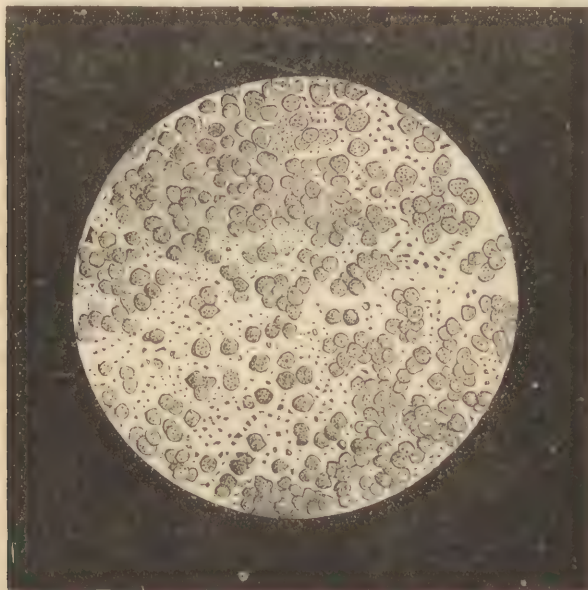
The same sputum after the addition of acetic acid.

homogeneous, and enable us to recognize two or more nuclei in the interior (Fig. 52).

The sputum at the beginning of a bronchial catarrh is the prototype of a mucous sputum, and its remarkable toughness and stickiness are due to the large amount of mucin it contains.

If the bronchial catarrh has lasted for some time, the sputum becomes more fluid, and expectoration is correspondingly easier. The proportion of mucin diminishes, that of water increases, and at the same time the sputum grows more profuse. It also loses its colorless, transparent appearance. At first it contains grayish-green opaque specks, from the size of a pin's head to that of a pea, but these grow larger and finally form yellowish-green balls of pus which float in a trans-

FIG. 53.



Muco-purulent sputum in acute catarrh of the large bronchi.

parent, muco-watery substance. This change in the sputum depends on the abundant admixture with pus-cells (Fig. 53). Thus the originally mucous sputum has become muco-purulent, and the cough is loosened. As the bronchitis subsides the sputum diminishes more and more in amount, and at the same time the desire to cough ceases.

The straining of the vocal cords often gives rise to hoarseness. Violent cough may also be accompanied by stitches in the side as a result of the increased tension of the expiratory muscles. The patients often complain of headache, dizziness, tinnitus aurium—the signs of venous congestion of the brain—on account of the disturbance produced by the cough in the domain of the superior vena cava. The veins of the neck may dilate into thick, bluish cords, and cyanosis may develop under such circumstances. Violent and repeated epistaxis is sometimes observed. The constant concussion experienced by the stomach during the coughing spells often gives rise to vomiting. Involuntary micturition

occurs not infrequently in old, feeble individuals, especially in females. Involuntary evacuations from the bowels are also observed occasionally. Violent coughing is especially dangerous in pregnant women and may be the cause of premature delivery. Hernia and prolapsus ani are also observed sometimes as sequelæ.

Acute catarrh of the large bronchi sometimes follows catarrh of the conjunctival, nasal, and laryngeal mucous membranes, sometimes it exists independently. It is usually bilateral, but may be unilateral, or confined merely to a single lobe.

If the catarrh is confined to the large bronchi, very little danger need be apprehended. The most serious symptoms are the result of the febrile movement, and this may give rise, in children, to cerebral symptoms, viz., somnolence, delirium, unconsciousness, localized spasms, and general convulsions. The disease usually lasts from a few days to one or two weeks.

The clinical history assumes a more serious character when the acute catarrh extends to the small bronchi (capillary bronchitis), particularly in children and old people.

The danger during childhood resides chiefly in the fact that the lumen of the small bronchi is unusually small in comparison with the alveolar space, so that relatively slight swelling of the mucous membrane, and accumulation of secretion will suffice to render the bronchi impenetrable to air. Furthermore, the inflammatory process in children presents an undoubted tendency to spread to the alveoli of the lungs and there give rise to secondary inflammation. The capillary bronchitis then terminates in catarrhal pneumonia (broncho-pneumonia).

The conditions are somewhat different in old age. In very old, and also in feeble and cachectic individuals, capillary bronchitis is dangerous on account of the severe general symptoms, and the usually long duration of the disease. The disease is not infrequently associated with considerable fever, which the organism of the old man can no longer withstand, and his vital energies fail. Hence the expectoration ceases, the secretion accumulates in the smaller air passages and gives rise to death from suffocation.

Capillary bronchitis may develop as such from the beginning or—and this is more frequent—it follows catarrh of the large bronchi.

The disease sometimes develops in a latent manner, without noteworthy disturbance of the general condition. As a rule, however, febrile movement is present, particularly in children and old people. The disease sometimes begins suddenly with severe febrile initial symptoms. Repeated chilly sensations or slight rigors open the scene (eclamptic attacks may occur in children), and are followed by more or less elevation of temperature, lasting for several days. The fever follows no distinct type, but is generally remittent, *i. e.*, the evening temperature is high, but the morning remission sinks to the normal. A temperature above  $39^{\circ}$  C. does not point positively to a complication with catarrhal pneumonia, but a temperature which continues at  $40^{\circ}$  C. for several days should arouse our suspicions. The course of the disease is most apt to be apyrexial during the period of vigorous manhood.

The signs of disturbed interchange of gases in the lungs are among the most important symptoms of capillary bronchitis. If these are associated with non-consonant fine râles, the diagnosis is certain.

Upon inspection, the frequency of the respirations is found to be



increased. In children the respirations often assume a hurried, panting character.

The expiratory respiratory murmur is not infrequently prolonged, and is accompanied, particularly in children, by a groan or moan. In rarer cases, inspiration is slow and prolonged.

If the distribution of the bronchial catarrh is unequal, the respiratory movements are also unequal, being delayed over those portions of the thorax which are most affected.

Inspiratory retraction of the thorax is a very important sign of disturbance of the pulmonary interchange of gases. In adults the retraction is confined to the intercostal spaces; but in children in whom the thoracic skeleton is yielding, the costal cartilages, ribs, ensiform cartilage, and epigastrium, take part, so that both hypochondriac regions and the epigastrium are drawn deeply inwards. This symptom shows that the entrance of air into the alveoli is prevented, so that when the thorax is dilated during inspiration, but the lung cannot take part in the inspiratory dilatation, the yielding portions of the thorax are forced inwards by the external atmospheric pressure.

As a rule, the inspiratory retractions first appear in the lower intercostal spaces because, as the result of stasis, the bronchitis is usually most severe in that region. But it must be remembered that some individuals present normally a sort of inspiratory retraction of the lower intercostal spaces. This is observed, however, only at the beginning of inspiration. The more the bronchitis spreads, the more the inspiratory retraction extends. It is rare that local and circumscribed retractions are seen. This occurs when the bronchiolitis affects only a small portion of the bronchial tree.

It is sometimes found in children that the lower parts of the thorax are retracted during each inspiration, but the upper parts are remarkably prominent anteriorly, and quite immobile. This is an important sign of acute distention and permanent inspiratory dilatation of the anterior portions of the lungs. This is owing to the fact that during inspiration the air passes through the blocked bronchioles into the alveoli, but cannot escape from the latter during expiration. As a matter of course, the alveoli gradually dilate and finally prevent even the inspiratory entrance of air.

In marked disturbance of the pulmonary interchange of gases the auxiliary muscles of respiration are brought into play. The *alae nasi* dilate shortly before the beginning of inspiration, the head is often drawn backwards and the mouth kept wide open, so that the respiration assumes a gasping character. At each inspiration, the larynx descends and draws the jugular fossa inwards. The sterno-mastoids, scaleni, and pectorals are brought into play during inspiration.

Auxiliary muscles are also employed in expiration, particularly the *recti* and *transversi abdominis*.

Certain positions of the body are often assumed in such conditions. In adults we observe an elevated dorsal decubitus or marked orthopnoea. In children, the dyspnoea increases if they are placed on the back and they instinctively ask to be carried about.

The excess of carbonic acid in the blood, and perhaps to a still greater extent the diminution of oxygen, are shown by the cyanosis. This occurs earliest in the face, particularly the cheeks, lips, conjunctiva, tip of the nose, and concha of the ears. It must be remembered that the disturbed interchange of gases in the lungs is not the sole cause

of the imperfect decarbonization of the blood, but that the flow of venous blood to the heart, which is aided normally by the aspirating and propulsive action of the lungs, is interfered with by the accumulation of secretion in the air passages and by the frequent coughing spells. When the venous circulation is obstructed, the veins of the neck are swollen, particularly after a spell of coughing.

If the carbonic acid poisoning assumes the upper hand, the skin often has an ashen-gray, livid appearance. Consciousness becomes clouded, the patients grow delirious, the eyes are closed, pupils contracted, muscular twitchings appear, and the patients die from suffocation. Cheyne-Stokes respiration is observed in rare cases.

Palpation of the thorax does not furnish any important data. The chest is not infrequently tender on pressure, particularly if the thoracic muscles have been strained by constant and violent coughing. The abdomen may also be very sensitive on the slightest touch. Vocal fremitus is not infrequently abolished, or very much diminished in certain parts, as soon as a large bronchial tube is occluded by an accumulation of secretion. The vocal fremitus reappears as soon as the secretion has been expectorated by coughing. In rare cases we can feel a peculiar crepitation and fine humming which correspond to the fine râles heard on auscultation. Bronchial fremitus may appear if catarrh of the large bronchi is associated with the capillary bronchitis.

Simple capillary bronchitis gives rise to no changes on percussion. Abnormalities in percussion always indicate the presence of complications. Thus, it is sometimes found that the borders of the lungs move downwards and towards the median line. This occurs when the median and lower borders of the lungs have undergone acute dilatation. Circumscribed areas of dullness, which are recognizable only on feeble percussion, are occasionally noticed. They may disappear if the patient takes long breaths or changes the position of the body for some time. They have been attributed to atelectasis, which is capable of complete restitution when the affected parts of the lung are forced to take an active part in respiration. If the dullness persists, it is an indication of complicating broncho-pneumonia.

The absence of dullness does not prove that spots of atelectasis or broncho-pneumonia are not present, for these may be too small to produce dullness, although they may be present in not inconsiderable numbers.

Auscultation furnishes the most important diagnostic signs. If the terminations of the bronchi are filled with secretion, the conditions are furnished for the production of fine moist râles, inasmuch as the walls of the bronchi are separated from the secretion during inspiration. The crepitant râles of simple bronchiolitis have no consonance, which is only present when there are cavities in the lung, or the pulmonary parenchyma is destitute of air. The respiratory murmur always remains vesicular in character, since the development of bronchial breathing requires the same conditions as the consonance of the râles. The respiratory murmur is sometimes absent in certain places if individual bronchi have become impervious. The bronchophony, though usually unchanged, is sometimes abolished or diminished. Catarrh of the finer bronchi may also give rise to interruption of the vesicular respiratory murmur, inasmuch as the transmission of sound varies within the bronchi, which are swollen to an unequal extent.

If bronchial breathing develops during capillary bronchitis; if the

râles acquire consonance; if the bronchophony is increased, we may assume the presence of collapse of the lungs, or broncho-pneumonia.

As a rule, the auscultatory signs of the bronchial catarrh and its complications are more marked in the lower, posterior portions of the lung. This is particularly true of children in whom, if these parts are free from change, we may be tolerably certain that no morbid conditions of any moment will be found in other parts. Nurslings should be examined in the sitting position.

The mothers have a habit of placing the children on the chest and abdomen, in order to facilitate the examination of the posterior surface of the chest. In this position, the little ones can breathe very little with the anterior portions of the lungs, which are burdened by the weight of the body, and, as the posterior portions take little part in respiration on account of disease, the patient soon suffers from dyspnoea.

Cough is always present in catarrh of the finer bronchi, but is not so violent as when the inflammation extends, at the same time, to the larger bronchi. The remarks made concerning the cough of catarrh of the large bronchi also hold good concerning the cough of capillary bronchitis.

At the onset of capillary bronchitis, the sputum is scanty, tough, transparent; later, it becomes more profuse, and contains the yellowish-green clumps of pus described before. It is evident from the peculiar formation of the sputum that it is derived, in part, from the small bronchi. If the sputum is placed in water, the frothy, muco-purulent clumps which are mixed with air bubbles, and are derived from the large bronchi, remain upon the surface. The secretion from the finer bronchi is destitute of air, and has a tendency to sink to the bottom, but it remains adherent to the upper portions, and therefore floats to and fro in the shape of fine, opaque threads which, to a certain extent, form a mould of the finer bronchi. It should be remembered that children under the age of seven years usually swallow the sputum.

Other organs may not remain unaffected in capillary bronchitis, but, as a rule, the implication of other organs does not possess any great significance.

The cardiac dulness may be either diminished or increased in size. Diminution of dulness indicates distention of the median anterior borders of the lungs, which encroach upon the anterior surface of the heart. The area of greater (relative) cardiac dulness sometimes extends outwards beyond the right border of the sternum. This change indicates dilatation of the right heart, and is the result of the impediment to the circulation of blood in the pulmonary artery produced by extensive catarrh.

In such cases, the volume of the liver is increased because the circulatory disturbance in the inferior vena cava acts upon the circulation in the hepatic veins.

Anorexia and increased thirst are usually the symptoms of the fever, while vomiting and irregularity in the evacuations from the bowels are the results of the cough and the abnormal distribution of blood. The abdominal walls are sometimes very sensitive to pressure, particularly when the cough is very violent or the recti and transversi abdominis act as expiratory muscles. In both cases, the pain must be attributed to strain of the abdominal muscles.

The pulse is usually much more rapid than would be supposed from the intensity of the fever. It beats not infrequently more than one hundred times a minute. If the disease ends fatally, the pulse usually can-



not be counted, and is almost imperceptible. At the same time, it becomes irregular.

The disease often lasts only a few days, but sometimes it extends over several weeks. Remissions and exacerbations are observed not infrequently.

Chronic bronchial catarrh may develop as such from the start, or it may follow relapsing acute catarrh. The etiology determines in part the character of the catarrh. Thus, catarrh due to cold is usually acute, that resulting from inhalation of dust or circulatory disturbances is not infrequently chronic from the beginning. Chronic catarrh is rare in children, but common at an advanced age.

In many patients the symptoms of chronic catarrh continue permanently. At times they increase in violence, particularly during the spring and autumn, evidently as the result of temperature changes. In other patients the signs of bronchial catarrh are present only at certain seasons. In this category is included that form of chronic bronchial catarrh known as winter cough.

The disease, as a rule, runs an apyrexial course unless violent acute exacerbations develop.

In the majority of cases chronic catarrh affects the large and medium-sized bronchi, so that creaking and sibilant râles, and coarse and moderately fine moist râles constitute the principal symptoms. It is only during acute exacerbations that the catarrh has a tendency to extend to the smaller bronchi, sibilant râles become prominent and fine râles also make their appearance. The identity of the local symptoms with those of acute bronchial catarrh is owing to the fact that they are dependent on mechanical and physical changes in the air passages. Any deviations which may appear are the result of the chronic character of the disease.

For example, considerable hypertrophy of the flexors of the head occurs in many cases because these muscles are constantly employed in the movements of respiration. Furthermore, the dilatation of the cervical veins is usually more marked than in acute bronchial catarrh. Chronic catarrh sometimes results in insufficiency of the valves of the internal jugular and crural veins. This condition is recognized by the fact that, during coughing spells, perceptible systolic thrills appear over the bulb of the internal jugulars (between the origins of the sternomastoid) and below Poupart's ligament. On auscultation, the thrills are found to correspond to systolic murmurs. This phenomenon is the result of regurgitation of blood from the heart through the insufficient valves of the veins. These conditions may also give rise to true venous pulse. More frequent than true venous pulse are the undulatory movements or respiratory variations in volume of the cervical veins. The former are synchronous with the movements of the heart, the latter appear as distention during expiration and coughing, and as diminution in size during inspiration.

All these circulatory phenomena are the effects of obstruction to the flow of blood from the pulmonary artery, and the propagation of the stasis through the right auricle into the superior and inferior vena cavae. They occur so much more readily the more protracted and extensive the catarrh, and the greater the amount of change in the pulmonary parenchyma.

The circulatory disturbances in question will give rise to dilatation and hypertrophy of the right heart. This is shown by the extension of the right border of the greater (relative) cardiac dulness beyond the

right edge of the sternum (dilatation), and by intensification of the diastolic sound over the pulmonary artery (hypertrophy). Failure of the right heart to meet the increased demands made upon it is followed by œdema of the limbs, scanty diuresis, and albuminuria, dropsy of the serous cavities, and increase of the bronchial catarrh. There is also danger of degeneration of the heart muscle and of failure of the heart's action, since advanced age in itself furnishes a tendency to degenerative changes in the muscular substance of the heart; and in addition, this predisposition is increased by the excessive amount of work required of the heart. Finally, disturbances in the pulmonary interchange of gases have a tendency to produce fatty degeneration of the heart muscle, chiefly as the result of the diminution of the amount of oxygen in the blood.

In another series of cases a fatal termination ensues from a complication with inflammation of the lungs. In some cases the sputum becomes very profuse, the patients lose strength in consequence, they emaciate and finally succumb to pseudo- hectic symptoms (phthisis pituitosa). Dangerous hæmoptysis rarely occurs. It may be the result of violent exertion while coughing, or of erosion of large bronchial vessels from putrid decomposition of the bronchial secretion. In other cases apparent bronchial catarrh is followed by the signs of pulmonary phthisis. Finally, accidental complications may be the cause of death.

Bronchial dilatation (bronchiectasis) sometimes develops as a complication of the disease. In other cases the secretion decomposes and gives rise to putrid bronchitis which may be followed, in its turn, by gangrene of the lungs. In still other cases chronic bronchial catarrh forms the starting-point of alveolar emphysema.

The disease often lasts many years, or for a lifetime.

The character of the sputum varies to such an extent as to give rise to special forms of disease:

*a. Dry bronchial catarrh* is characterized by a very scanty, tough, vitreous gray secretion. It occurs most frequently at an advanced age. The cough is apt to be particularly constant and severe. It appears not infrequently in paroxysms, or attacks of dyspnoea occur which are entirely like those of asthma. This form of disease very often gives rise to emphysema.

*b. Bronchorrhœa simplex* is characterized by a very profuse muco-purulent sputum. If the mucous and watery constituents are abundant, round balls of opaque, greenish pus are formed. These sink in part, but in part are kept up by bubbles of air. Very large masses are sometimes expectorated, even though the local changes are not very marked. We not infrequently see patients who expectorate several pounds of fluid in a few hours, although very little or no change can be demonstrated in the lungs. Such conditions may continue unchanged for years. Signs of increasing emaciation gradually become noticeable.

*c. In bronchorrhœa serosa* the secretion is very profuse, extremely thin, colorless, transparent, frothy, and extremely poor in cells. Its expectoration requires very violent coughing spells, which may be followed by rupture of small vessels in the bronchial mucous membrane. Asthma-like attacks often occur suddenly and disappear after large amounts of secretion have been expectorated (asthma humidum). The patients often withstand the disease for a very long time. Laennec mentions the case of a patient, who enjoyed tolerable general health, despite the fact that for ten to twelve years he had expectorated daily about four pounds of sputum.

d. In *broncho-blennorrhœa* the sputum is almost entirely purulent. In addition to pus-cells, the sputum contains fatty cells, and fatty and granular detritus. The absence of shreds of lung tissue and elastic fibres readily distinguishes this condition from pulmonary abscess, and the absence of pleurisy, pericarditis, or suppuration in the abdominal cavity testifies against the perforation of the pus into the lungs from the outside.

e. The secretion of simple chronic bronchial catarrh sometimes undergoes putrid decomposition (putrid bronchitis). The sputum has a cadaverous stench, which has also been likened to horse-radish or garlic. Some patients emit such a stench that they are obnoxious to themselves and to those around them. The foul odor of the expired air should not be mistaken for *factor ex ore*. As a rule, the latter is only noticeable if the nose is held in front of the patient's mouth, but the factor of putrid bronchitis is undiminished at a few paces from the patient. On account of this stench the patients not infrequently have an unconquerable antipathy against food. In many cases, however, the bad odor is only temporary. It is very distinct in the fresh sputum, but disappears after the sputum has been standing in the open air for some time, and reappears only when it is thoroughly shaken.

The amount of the sputum is usually very considerable (two hundred to five hundred ccm. or more). It is generally a thin, grayish-green fluid which is occasionally streaked with blood, or looks clay-colored from changes in the blood pigment. It has a characteristic tendency to separate, after long standing, into three (more properly four) layers. The lowermost one is a sediment; it is granular, grayish-green, ashen, or brownish-gray. The middle layer is usually a thin, watery fluid. The upper layer is very frothy, and also contains muco-purulent clumps.

In 1850, Dittrich discovered in the sedimentary layer peculiar plugs (mykotic bronchial plugs, Dittrich's plugs) which were proven by Traube to be characteristic of putrid bronchitis. Their size varies from that of a pin's head to that of a finger nail. They are whitish, grayish, or brownish in color; the whiter they are, the more recent is their development. Their consistence is like that of porridge, and on being squeezed they emit an extremely foul stench.

In the main they consist of granular detritus which, under sufficiently high powers, is resolved into fine, short, occasionally geniculated threads, and into extremely small round spores (*leptothrix pulmonalis*). Upon the addition of tincture of iodine, this fungus assumes a brownish-yellow, violet-blue, or purple-violet, and even blue color. The plugs also contain spirilli which are recognized by their graceful, corkscrew-like twists.

In addition to schizomycetes, the recent plugs also contain pus-corpuscles. In the older plugs cellular elements are almost entirely absent. At first more or less numerous drops of fat appear, then a few short, fine needles of margaric acid; later, these needles become more profuse, thicker, longer, and are arranged in tufts or in delicately curved lines. More or less changed red blood-globules, flakes of pigment, and even hæmatoidin crystals are sometimes found.

If the needles of the fatty acids are densely aggregated and have a very winding course, there is danger of mistaking them for elastic fibres. But the latter have a more distinct double contour, and not infrequently branch dichotomously. The needles of margaric acid, on the other hand, are dissolved in ether, alcohol, and the caustic alkalies, and have a tend-



ency to melt upon being warmed. On pressure they present varicose enlargements.

Jaffé found that the putrid sputum contained volatile fatty acids, particularly butyric and valerianic acids, leucin, tyrosin, traces of glycerin, occasionally sulphuretted hydrogen, and ammonia. From the plugs he obtained a white, brittle substance, which turned blue on the addition of iodine, but was neither amylum nor a protein substance. Filehne and Stollnikow obtained from the putrid sputum a ferment-like substance, similar in its action to the pancreatic ferment (trypsin).

It seems a very plausible theory that the decomposition of the sputum is effected by the low vegetable organisms in the plugs. Rosenstein observed a case in which a young girl inhaled *oidium albicans* from a patient in an adjoining bed, and, as a result, suffered from putrid bronchitis. Camali recently reported a case of putrid bronchitis following *aktinomykosis* of the bronchial passages.

The sputum of pulmonary gangrene is similar to that of putrid bronchitis, except that the former contains shreds of the parenchyma of the lungs. Putrid bronchitis sometimes is gradually converted into pulmonary gangrene, inasmuch as the sputum erodes the walls of the bronchi, and the process of destruction and decomposition then extends to the substance of the lung. Putrid bronchitis is not always preceded by the development of bronchiectasis. It is true that the stagnation of secretion in the latter favors putrid decomposition, but bronchiectasis is not a necessary condition of the development of putrid bronchitis.

Patients suffering from the latter disease often present a healthy complexion for a very long time. But if the disease has lasted a long time, signs of emaciation appear, particularly if the expectoration of the secretion is sometimes checked. Suppurative fever develops, hemorrhages occur, the appetite is lost. Obstinate diarrhoea is observed occasionally, and is perhaps the result, in part, of ingestion of the putrid sputum by the patient.

IV. DIAGNOSIS.—The diagnosis of bronchial catarrh is not always easy. There can be very little question if we detect non-consonant râles, feeble respiratory murmur, intensified, jerky vesicular breathing, prolonged expiration, absence of dullness. The site of the catarrh is determined by the character of the râles. Creaking and coarse mucous râles indicate catarrh of the large tubes; hissing and whistling indicate catarrh of the finer tubes; fine subcrepitant râles and jerky breathing, catarrh of the capillary bronchi. If the secretion is tough, the râles are dry; if the secretion is fluid, the râles are moist.

The absence of all resonant phenomena (bronchial breathing, consonant or metallic consonant râles, increased bronchophony, increased vocal fremitus) distinguishes bronchial catarrh from pneumonic processes. Furthermore, dullness appears in the latter if the inflammation assumes certain dimensions.

When creaking râles and muscular thoracic pains are present, the diagnosis from dry pleurisy may be doubtful. But if the patient coughs, the secretion is often removed from the bronchi, and the creaking disappears or diminishes greatly, while a pleuritic friction murmur is unaffected under such circumstances. Moreover, pleuritic friction murmurs are often intensified upon pressing the stethoscope firmly into the intercostal spaces, while the râles remain unchanged.

V. PROGNOSIS.—In some cases, medical interference is hardly required; in others, the disease is very dangerous. The prognosis depends

chiefly on the age of the patient and the location of the catarrh. In children and old people, bronchial catarrh is always a serious affection, and the prognosis is especially grave when the smaller bronchi are affected. Complications must also be taken into consideration. For example, the prognosis is always grave when the catarrh is associated with pulmonary inflammation.

Acute bronchitis presents more chances of complete recovery than the chronic form. Certain complications of chronic catarrh, for example, bronchiectasis, are hardly susceptible of recovery.

VI. TREATMENT. — Prophylactic measures form an important part of treatment.

If the tendency to bronchitis is maintained by enervation, the body should be "hardened" in a rational manner. This is indicated particularly in children. But we must avoid proceeding either too rapidly or in a half-hearted manner. It would not profit much to have the children subjected to cold frictions, morning and evening, and then allow them to go improperly clothed in the open air in inclement weather. It is especially hurtful to keep the children in rooms which are still damp from a recent scrubbing.

Individuals who work in a dust-laden atmosphere should breathe through the nose, and wear respirators. Much may also be done by proper ventilation and improvement in the construction of factory buildings.

In certain cases, the treatment of the bronchitis coincides with that of the primary disease (rickets, scrofula, chlorosis, anæmia, marasmus). This is also true of toxic bronchitis, which disappears on the removal of the cause.

In catarrh of the large bronchi, which is annoying chiefly on account of the cough, we may prescribe narcotics in small doses, for example:

℞ Pulv. ipecac. opiat.,  
Sacch. alb. .... āā 0.3  
M. f. p., d. t. d. No. x. S. One powder t. i. d.

℞ Aq. amygdal. amar. .... 10.0  
Morphin. muriat. .... 0.1  
M. D. S. 10 drops t. i. d., or when the cough is annoying.

Narcotics must be given very cautiously in children, as they readily give rise to serious symptoms of poisoning.

If there are numerous sonorous and sibilant râles, we should endeavor to liquefy the secretion, and facilitate expectoration. Too much must not be expected from the expectorants (ammon. muriat., apomorphin. muriat., camphora trita), but the following prescriptions may be recommended:

℞ Ammon. muriat.,  
Succi liquiritiæ ..... āā 5.0  
Aq. .... q. s. ad 200.0  
M. D. S. One tablespoonful every hour or two.

℞ Apomorphin. muriat. .... 0.05  
Acid. muriat. .... 0.5  
Aq. .... 200.0

M. D. S. One tablespoonful every two hours.

℞ Inf. rad. ipecacuanh.....	1.0 : 180.0
Kali iodid. ....	3.0
Syrup. simp.....	20.0

M. D. S. One tablespoonful every two hours.

The patients usually experience great relief from careful regulation of the temperature of the room (15° R.), especially if the air is kept constantly moist. This is done readily in winter by placing vessels of water upon the stove. Siegle's inhalation apparatus, or an ordinary spray apparatus, may be employed for the same purpose, its contents being forced into the air of the room every hour or two. The great value of this measure in the capillary bronchitis of children has been recently shown by Abelin and Hansen, the former noting a reduction of the mortality in infants (in the Stockholm Children's Hospital) from 48 per cent to 18 per cent.

A still greater effect is obtained in many cases if the vapor of water is saturated with alkalies or balsams (solutions of chloride of sodium, bicarbonate of soda, carbonate of soda [1-5 per cent], Ems or Selters water, turpentine, carbolic acid [2 per cent], creasote [0.5-1 per cent], tar water [10-20 per cent], etc.)

A still more marked effect is obtained from direct inhalation of these substances (at least once every three hours). But the results of local treatment of bronchial catarrh by means of inhalation have not come up to our expectations, probably because the inhaled fluid does not pass beyond the trachea or primary bronchi.

If numerous râles are heard, it may be assumed that the bronchial secretion is fluid, and we must attempt, accordingly, to secure its mechanical removal. The following prescriptions may be recommended :

℞ Inf. rad. ipecac.....	0.5-1.0 : 100
Syrup simp.....	20.0

M. D. S. One tablespoonful every two hours.

℞ Inf. rad. ipecac.....	0.5-1.0 : 100
Aq. amygdal. amar.....	5.0
Syr. simp.....	15.0

M. D. S. One tablespoonful every two hours.

℞ Inf. rad. ipecac.....	0.3 : 100
Stibii sulphurat. aurantiac.....	0.3
Syr. simp.....	20.0

M. D. S. To be well shaken. One teaspoonful every two hours.

℞ Decoct. rad. senegæ....	10 : 180
Liq. ammon. anisat.....	5.0
Syr. simpl.....	15.0

M. D. S. One tablespoonful every two hours.

℞ Acid. benzoic.....	0.1
Ext. belladonnæ.....	0.01
Sacch. alb.....	0.5

M. f. p., d. t. d. No. x.

S. One powder to be taken every three hours.

If the air passages are overloaded with secretion, the administration of emetics is often indicated (ipecacuanha, tartar emetic, sulphate of copper, muriate of apomorphine, etc.). We should not delay until the



carbonic-acid poisoning has gained the upper hand, because the irritability of the pneumogastric centre is then diminished to such an extent that the emetics prove ineffective. For this reason the emetics should not be given repeatedly until emesis occurs, as symptoms of poisoning are produced not infrequently. The emetics often become effective if the irritability of the central nervous system is increased by giving a few teaspoonfuls, or in adults tablespoonfuls, of brandy or strong wine, half an hour before the administration of the emetic. As symptoms of collapse appear not infrequently after the use of emetics, we must be prepared, immediately after emesis, to give wine in large doses. The prescriptions mentioned on page 142 may be ordered.

The overloading of the air passages with secretion is sometimes owing to the fact that the patient is too weak to cough vigorously or that he falls into a somnolent condition as the result of carbonic-acid poisoning. In such cases we recommend stimulants: wine, camphor, benzoic acid, liq. ammon. anisat., musk, etc.

℞ Camphoræ tritæ.....0.05  
 Acid. benzoic.....0.3  
 Sacch. alb.....0.5

M. f. p. No. x. S. Take one powder every hour or two.

℞ Liq. ammon. anisat.....5.0

D. S. Five drops on sugar every hour.

℞ Moschi,  
 Sacch. alb.....āā 0.3

M. f. p. No. v. S. Take one powder every hour.

In diffuse bronchitis, of old people in particular, the treatment should be stimulating from the start, because adynamic symptoms are apt to develop. In many cases an extremely good effect is obtained from a warm bath, cold water being poured from a height upon the chest while the patient is in the bath. The patient takes a deep breath involuntarily, the air passages are often freed in this manner, and the carbonic acid narcosis may disappear.

The employment of pneumatic cabinets and portable pneumatic apparatus (Waldenberg, Geigel-Meyer) is often attended with decided and surprisingly rapid effects. They facilitate expectoration and recovery, not alone in chronic, but also in acute catarrh. If inspiration is obstructed (as is generally the case) compressed air should be employed; if expiration is remarkably prolonged and accompanied by abundant râles, the patient should expire into rarefied air. If both phases of respiration are impeded, compressed and rarefied air should be employed alternately. This should be done several times a day, each sitting lasting from half an hour to an hour.

Treatment by means of derivatives is often employed. A decided effect can be expected from diaphoretics only when acute bronchitis has resulted from a cold. Cathartics are employed particularly in children. The following is a favorite prescription.

℞ Calomelanos,  
 Stibii sulphurat. aurant.....āā 0.05  
 Sacch. alb.....0.5

M. f. p. No. x. D. S. Take one powder t. i. d.

Many patients suffering from chronic bronchial catarrh do very well at the seashore or recover permanently on taking a long sea voyage. Sool baths and alkaline and alkaline-muriatic waters may also be recommended. The earthy mineral springs sometimes prove beneficial. If the patients are puffy and bloated, we may recommend the springs of Kissingen, Marienbad, Carlsbad, Homburg, Wiesbaden, and Tarasp.

Uniformity of climate is the chief thing to be considered with regard to change of air. In summer, well wooded and protected mountainous regions may be recommended, in autumn the patients may be sent to Lake Geneva or the Tyrol, in winter to places on the Riviera or localities still further south.

Special indications must sometimes be met in the course of bronchial catarrh.

Warm compresses, mustard poultices, or dry cups are useful when the patients complain of violent pains in the chest. The most rapid and certain results are usually obtained by subcutaneous injections of morphine.

If high fever has developed, antipyretics are indicated. The most certain one is antipyrin, of which 3.0–5.0 dissolved in three tablespoonfuls of lukewarm water may be given by enema.

If hæmoptysis sets in, the patient should be kept absolutely quiet, pieces of ice may be given, and at the same time injections of ergotin and full doses of narcotics, for example :

R Plumbi acetic.....	0.05
Opii puri.....	0.03
Sacch. alb.....	0.5
M. f. p. No. x. S. Take a powder every two hours	

If asthmatic symptoms arise, we may give iodide of potassium for a long time; this remedy may be combined with infusion of ipecac. Narcotics may be given if the respiratory disturbance is very great.

In bronchorrhœa and broncho-blemnorrhœa, the greatest benefit is obtained from inhalations of balsams. They diminish the secretion and prevent its decomposition. The following prescription may be used :

R Ol. terebinthinæ,	
Aq.....	āā 50.0
Vitelli ovi I.	
Ol. lini.....	5.0

M. f. linimentum. D. S. One teaspoonful rubbed on the chest morning and evening; the bed clothes then drawn over the head, and deep inspirations taken.

Not much can be expected from the internal administration of as-tringents.

Putrid bronchitis requires the same treatment as pulmonary gan-grene. This will be described in a subsequent section.

## 2. *Fibrinous Bronchitis* (*bronchial croup, bronchitis crouposa s. pseudomembranacea.*)

I. **ETIOLOGY.**—In fibrinous bronchitis an exudation is formed which is rich in fibrin and forms casts of the bronchi. It may be either primary or secondary.

Secondary bronchial croup is inflammation which is propagated from adjacent parts. The inflammatory process either begins in the larynx

and spreads downwards, or it develops in the pulmonary alveoli and extends upwards. It is therefore found very often in laryngeal croup and fibrinous pneumonia. It may also complicate pulmonary phthisis, particularly towards the end of life.

Primary fibrinous bronchitis is an independent affection which begins in the bronchi. It is not always confined to the bronchi; it sometimes extends to the larynx, at other times spreads to the alveoli where it gives rise to fibrinous pneumonia.

Primary and secondary fibrinous bronchitis cannot always be sharply differentiated. Cases have been reported in which primary fibrinous pneumonia led to the development of secondary fibrinous bronchitis. The pneumonia then disappeared and the fibrinous bronchitis remained as an independent affection.

Primary bronchial croup is not a frequent disease. Hardly more than a hundred cases have been reported. It occurs most frequently from the age of 10 to 30 years; it is found with greater relative frequency in childhood than in old age. Hayn observed this lesion in the body of a new-born babe. Almost all writers agree that the disease is more frequent in the male sex. The majority of the patients are anæmic and feeble.

A few observations show that several members of one family may be affected with the disease, but hereditary influence has not been demonstrated with certainty.

The disease is rare in southern Europe, but seems to be especially frequent in Switzerland. It occurs particularly in the months of May and June.

Very little is known concerning the immediate causes of bronchial croup. It is not infrequently the result of a cold. A simple bronchial catarrh first appears and then passes into bronchial croup; more rarely the latter affection develops as such from the start.

Cohnheim states that cheesy lymphatic glands, which have ruptured into the trachea or bronchi, may give rise to fibrinous inflammation.

Pregnancy and menstruation have been known to exert a certain effect in some cases. Oppolzer and Schnitzler report a case in which fibrinous bronchitis always developed at the menstrual period.

Eisenlohr described a case in which bronchial croup, which had been preceded by catarrhal bronchitis, developed during the second week of typhoid fever. In Jaeger's case, the disease began a few days after recovery from measles. Gerhardt observed the disease in a young woman who suffered from valvular lesion of the heart.

Attention has been called recently to the relations between bronchial croup and diseases of the skin. Waldenburg observed bronchial croup in a child suffering from an impetiginous eruption on the scalp. In Streets' case, herpes zoster appeared upon the back of the neck and the shoulder during the attacks of croup; after the bronchitis subsided, an impetiginous eruption made its appearance. In Escherich's case, the fibrinous bronchitis was preceded by herpes of the lips, tongue, and pharynx. Mader noticed the association of fibrinous bronchitis with pemphigus of the mouth, nose, and conjunctiva.

II. SYMPTOMS.—Fibrinous bronchitis may run an acute or chronic course. Acute bronchial croup lasts a few days, rarely more than two weeks; the chronic form may continue for months and even years (14 years in Walshe's case). As a rule, its course presents exacerbations and remissions, the intervals of good health sometimes lasting for months.



Acute bronchial croup is much more dangerous than the chronic form. This is partly owing to the fact that the acute form has a great tendency to spread to many bronchi and also to the larynx. It also occurs much more frequently in the large bronchi, while chronic fibrinous bronchitis develops usually in the bronchi of the third and fourth orders, and presents a tendency to spread to the alveoli.

The two principal symptoms are alike in acute and chronic fibrinous bronchitis. The most important one is the expectoration of branching fibrinous clots, which form casts of the bronchial ramifications. Previous to the expectoration of the casts, there are signs of bronchial occlusion or stenosis, and these may lead to a fatal termination if they develop very rapidly or affect a large part of the bronchial tree. Death sometimes occurs before the bronchial casts are expectorated.

Fibrinous bronchitis rarely begins suddenly. In the majority of cases it is preceded by symptoms of bronchial catarrh, which begins occasionally with repeated chills and with febrile movement. Hæmoptysis occurs not infrequently in this prodromal stage and may assume threatening dimensions. More or less severe hæmoptysis sometimes occurs during or immediately after the expectoration of the bronchial casts. The disease is sometimes preceded by symptoms of angina.

The transition of catarrhal into fibrinous inflammation is often recognized by the occurrence of a chill with subsequent considerable elevation of temperature. After the expectoration of bronchial casts, the fever subsides, but the development of fresh casts is often preceded by another chill and rise of temperature.

While the fibrinous exudation is progressing, the signs of broncho-stenosis make their appearance. The patients suffer from dyspnoea and fear of suffocation. Many complain of a feeling of pressure and pain in the chest. The frequency of respiration increases, and the auxiliary muscles take part in the respiratory movements. The pulse grows frequent and tense. Signs of cyanosis appear in the face. Circumscribed retraction of the thorax may be observed or, if the fibrinous inflammation extends into a main bronchus, a large part of the thorax may take very little or no part in the respiratory movements.

On palpation, vocal fremitus is found to be diminished or abolished over the region of the affected bronchi (interference in the obstructed bronchi with the conduction of the waves of sound from the larynx to the chest walls).

The respiratory murmur disappears over the region of the occluded bronchi, but the percussion note is unchanged. Dulness will be present only when the fibrinous process has extended to the alveoli and has given rise to fibrinous pneumonia or when collapse of the lung has occurred from absorption of the air in the alveoli situated on the peripheral side of the occluded bronchi.

The expectoration of the bronchial casts is attended usually with very violent cough, more rarely with only slight cough. It is expectorated occasionally after an attack of vomiting. If the casts are very large, they may give rise to marked asphyxia during expectoration on account of the occlusion of the larger bronchi. A few hours generally elapse between the first symptoms which indicate the formation of the casts and their final expectoration. The attacks are generally more prolonged in chronic than in acute cases.

In some cases, small casts are found in a mucous or muco-purulent sputum, in others the casts predominate and there is only a slight ad-

mixture of mucus, pus, or blood. Only one or two casts may be expectorated in the course of a day, or their formation may be more active. They sometimes manifest a certain degree of periodicity. Puchelt describes a case in which the casts were expectorated in such quantities that they burst from the mouth and nose, and filled glasses of considerable size.

FIG. 54.

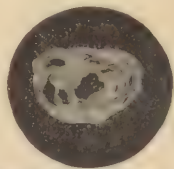


Bronchial cast from the sputum of primary chronic bronchial croup. From a dry preparation spread on glass. Natural size.

The casts almost always appear in the shape of rolled-up coils, which, if considerable blood is adherent to them, look like a mass of flesh. In other cases, they have a white or dull gray color, the latter color being produced by the blackish pigment cells contained in them. When stirred in water, they are resolved into branching cylinders, the ramifications of which enable us to recognize the shape of the bronchial tree (Fig. 54). On being shaken in water, the flesh-colored clots soon lose their color, and become white.

The length of the casts varies greatly, in some cases exceeding 18 cm. A girl of 15 years, who was treated by Niemeyer, expectorated daily a complete cast of the left bronchial tree.

FIG. 55.



Transverse section of a large bronchial cast. Double the natural size.

The main trunk may be as thick as a lead pencil or even the little finger. The casts in the large bronchial tubes appear not infrequently to be flattened, while the finer ones are twisted occasionally in the shape of a spiral. The beginning of the cast is not infrequently attenuated into a cone. The finer ramifications often contain vesicle-like dilatations which correspond generally to collections of air. The finest ends are either pointed or swollen into little knobs (casts of the infundibula).

The trunk and main branches are usually hollow and contain mucus and pus, the finer ones are almost always solid. The central axis is sometimes black and composed of numerous pigment cells.

Under the microscope the casts are found to have a lamellated

FIG. 56.



Transverse section of the wall of a fibrinous bronchial cast. Enlarged 275 times.

structure, the individual lamellæ being arranged either concentrically or in irregular layers (Fig. 55). The number of lamellæ varies, but their arrangement points to exudation by fits and starts.

The casts consist, in the main, of hyaline basement substance which in places is arranged in streaks (Fig. 56). The periphery often contains red blood-globules, sometimes in a condition of beginning degeneration.

In a case described by Flint, granules and crystals of hæmatoidin were present. A few more or less fatty round cells and fatty granules may be noticed. Flint also mentions the presence of pavement epithelium cells, but these probably adhered to the casts in their passage through the mouth. On the addition of lime-water, the basement substance dissolves and the cellular elements are set free. Acetic acid produces swelling of the basement substance. Colorless crystals in the shape of double pyramids are found not infrequently in the casts. They are entirely similar



to the Charcot-Neuman crystals or Leyden's asthma crystals (vide Fig. 58). The casts are sometimes destitute of all formed elements. Escherich observed one case in which the sputum became green after standing. In addition to the fibrinous clots, he also found spirals, which are depicted on page 232.

After the expectoration of the bronchial casts, the dyspnoea rapidly subsides and the patient feels considerably relieved. At the same time the respiratory movements become slower and freer. All parts of the thorax take part in respiration. The pulmonary vesicular murmur and vocal fremitus reappear. Whistling or fine moist râles are often heard over the locality which was previously affected.

Spaeth showed in one case that the vital capacity of the lungs was increased. Before the expectoration of the casts it amounted to 1,317 ccm., after expectoration it increased to 1,675 ccm.

The disease sometimes ends with the single attack, but, as a rule, the attacks recur several times daily for days and weeks. The disease is sometimes prolonged for several years. The expectorated casts often present an extraordinary similarity to one another, so that they are evidently derived from the same part of the bronchial tree.

In many cases the bodily vigor suffers very little even if the disease is chronic. Pleurisy, collapse, and retraction of the lungs, acute dilatation and emphysema of the lungs may occur as complications.

III. ANATOMICAL CHANGES.—The anatomical diagnosis is readily made if fibrinous clots are found in the bronchi. They are generally separated from the bronchial mucous membrane by air or mucus. In the finest bronchi they occasionally have a creamy consistence, so that they may be mistaken for inspissated mucus. The latter shrink on the addition of acetic acid.

The bronchial mucous membrane is generally reddened and swollen, and sometimes contains small hemorrhages. More rarely the mucous membrane is extremely pale. The epithelium of the mucous membrane beneath the casts is sometimes intact, sometimes it is entirely destroyed.

IV. DIAGNOSIS.—The diagnosis is easy if the casts are expectorated. When this does not happen, the diagnosis remains doubtful. It must then depend upon the acute development of the signs of broncho-stenosis and the exclusion of other causes (foreign bodies, retracting cicatrices, compression from the outside).

V. PROGNOSIS.—The prognosis of acute fibrinous bronchitis should be given with great caution. The disease has a tendency to a fatal termination, and death from asphyxia often occurs with unexpected rapidity. In chronic fibrinous bronchitis, also, the prognosis is not favorable.

VI. TREATMENT.—The loosening of the casts is aided by inhalations of the vapor of water, either directly, or indirectly by spraying the atmosphere of the room. Inhalations of lime-water, the alkaline carbonates, and lactic acid have also been recommended, but these remedies do not appear to possess any advantages over the vapor of water.

Inhalations of five-per-cent solutions of papayotin, neurin, tetramethylammoniumhydroxyd and tetraethylammoniumhydroxyd have been recommended as specially vigorous solvents of fibrinous clots.

If we have reason to believe that the casts are loosened, we should prescribe expectorants or, in vigorous persons, emetics. Among the

expectorants we may particularly recommend: ipecac, senega, liq. ammon. anisat., benzoic acid, and among emetics apomorphine.

The new-formation of fibrinous casts in acute bronchial croup has been combated by the use of mercurial preparations (vigorous inunctions with blue ointment until salivation begins, calomel, or corrosive sublimate internally). Iodide of potassium (10 : 200, one tablespoonful t. i. d.) has been successfully employed in several cases of chronic fibrinous bronchitis.

### 3. *Bronchial Asthma.*

(*Bronchial spasm. Asthma convulsivum.*)

**I. ETIOLOGY.**—Bronchial asthma consists of attacks of dyspnoea resulting from spasmodic contraction of the bronchial muscles. It is assumed that the smooth muscular fibres of the bronchi are innervated by the pneumogastric and spinal accessory, and as the disease presents no tangible anatomical lesions, it may be termed a neurosis of the accessorio-vagus.

Age exerts some influence on the development of bronchial asthma. It is observed most frequently between the 20th to the 40th years, and is much rarer after the age of 40. It is not very rare in childhood, and has been observed before the end of the first year. Indeed, Hyde Salter claims that the majority of cases begin during the first ten years of life.

The male sex predominates. Among 153 cases collected by Salter, 102 were males and 51 females.

In the third decennium females are attacked by this disease more frequently than males.

Rachitic, scrofulous, anæmic, and nervous individuals appear to have a special predisposition to the disease.

In some cases the influence of heredity appears to come into play, but its importance should not be overestimated.

Climate and the seasons affect certain forms of bronchial asthma, inasmuch as cold, changeable weather predisposes to the development of bronchitis and thus to the production of symptomatic bronchial asthma.

The disease is more frequent among the well-to-do than among the poor.

From an etiological standpoint the disease is divided into two classes, the idiopathic (essential) and the symptomatic (reflex). The causes of the former variety cannot be ascertained, those of the latter are situated in the other organs. This etiological relation becomes especially evident in those cases in which the cure of the primary affection likewise produces relief of the secondary bronchial asthma.

In rarer cases the trunk of the pneumogastric is affected, either at its central origin in the medulla or in its peripheral course.

Jolly and Ollivier have described cases of asthma in which lesions were found in the brain and spinal cord, but recent writers have criticised these statements and maintain that the occurrence of central bronchial asthma is, to say the least, unproven.

A number of cases have been reported in which asthma was produced by tumors which compressed and irritated the trunk of the pneumo-

gastric (lymphatic glands, enlarged thyroid gland, swollen tracheo-bronchial glands). The latter lesion plays a prominent part in the bronchial asthma of children, and may generally be assumed to be present when children suffer from asthma after measles, scarlatina, whooping-cough, etc. Scrofula and rickets should also be taken into consideration as causal factors. Eisenschuetz reports a case in which a child, born with enlargement of the thyroid (as large as a hen's egg), suffered from asthma. At the autopsy, it was found that the goitre had not produced stenosis of the trachea.

In certain cases, the reflex irritation starts from the nasal or nasopharyngeal cavity. A number of cases have been reported in which naso-pharyngeal polypi gave rise to asthma, the latter disappearing on the removal of the tumor, and reappearing when the neoplasm returned. Bronchial asthma is much more frequent when the mucous membrane of the inferior and middle turbinated bones is pathologically congested. Mackenzie has observed asthma as a result of chronic atrophic rhinitis, and has seen the former disease disappear when the nasal affection was relieved. Hypertrophy of the tonsils may also give rise to asthma, and in three cases I have seen the asthmatic attacks disappear after extirpation of the tonsils.

In some individuals, certain smells, such as perfumes, chlorine, coffee, etc., constitute a cause of asthma. Trousseau relates that in himself the odor of violets always produced asthma. We must carefully differentiate those cases in which certain odors constitute the exciting cause of an attack in individuals predisposed to bronchial asthma, from those in which asthma never occurs except as the result of the smell in question.

In some cases, the attack is the result of a mechanical irritation of the nasal mucous membrane (inhalation of powdered ipecac root, the dust of oat, hemp or corn straw).

Bronchial asthma occurs not infrequently as a complication of bronchitis, but we must be careful not to mistake dyspnoea for asthma. Asthmatic attacks also occur in diseases of the heart.

Diseases of the abdominal organs furnish a fertile field for the production of asthmatic attacks.

It may be produced by distention or by abnormal processes of fermentation in the stomach. Some individuals suffer from asthma after the ingestion of certain articles of food. Asthma may be the result of constipation or helminthiasis. It is observed very often in uterine and ovarian affections. Even pregnancy gives rise occasionally to bronchial asthma.

In diseases of the kidneys, asthma may appear as a part of the clinical history of uræmia (asthma uræmicum), evidently as the result of blood poisoning with urea (and other excrementitious substances). In the cases which I have seen, the patients almost always exhaled a urinous odor from the mouth. To this, to a certain extent toxic form of asthma, may be added that form which is the effect of lead and mercurial poisoning. Those cases in which attacks of gout alternate with asthma probably belong to this category.

An etiological relation is said to have been observed in some cases between cutaneous eruptions and bronchial asthma.

It is said that the disease may occur after recovery from chronic eczema. Raynaud and Brigault maintain that it sometimes develops as



the result of urticaria, but it is more probable that both affections are the result of a "spoiled" stomach.

II. ANATOMICAL CHANGES.—We have already said that asthma is a neurosis of the *accessorio-vagus*; in other words, no palpable lesion is found after death. When it is reflex in its origin, the primary organic affection will be discovered. We may also call attention to the fact that bronchial asthma may give rise to secondary changes in the lungs, viz., acute dilatation, emphysema, and atelectasis.

III SYMPTOMS.—The clinical history of bronchial asthma is characterized by attacks of dyspnoea of the expiratory type, associated with signs of acute dilatation of the lungs.

These attacks may occur suddenly, or they are preceded by prodromata, such as general malaise, a feeling of pressure in the back of the head or the forehead, yawning, eructations, vomiting, irregular stools, distention of the abdomen, slight chilly feelings, etc. Some patients know that an attack will occur as soon as they expose themselves to a certain noxious influence. The exciting causes may be very trivial, such as sleeping in a dark room or with closed doors, if they have been accustomed to the opposite condition. In some, the prodromata consist of conjunctivitis and coryza, which rapidly extend downwards, attack the bronchi, and lead to a seizure of asthma. The appearance of the menses also occasionally gives rise to bronchial asthma.

The attacks begin most frequently at night, particularly during the first few hours after midnight. The attacks occur occasionally at certain hours and on certain days, so that we are reminded of latent intermittent fever.

When the seizure occurs at night, the patients are aroused suddenly from sleep with a feeling as if they were choking. This sensation increases rapidly in intensity, and the more violent the respiratory movements, the greater the dyspnoea becomes. Many patients rush at once to the window to let fresh air into the room, since this affords some relief. Wheezing sounds in the chest are soon produced, and may be so loud as to be heard in a number of rooms. In certain cases, involuntary micturition and defecation occur during the attack. After the patients have thought that death was impending, respiration gradually grows freer. A slight cough with muco-purulent expectoration is usually observed. The wheezing ceases, and respiration no longer requires abnormal muscular effort. Yawning, eructations, or vomiting are noticed in some cases, and then respiration becomes normal.

The attack may last from a few minutes to one, two, or more hours. The disease sometimes terminates with a single attack. Some patients suffer from one or more attacks a day, but there are usually intervals of weeks or months in which they are free from the disease.

The following are the objective changes in an attack of asthma :

The patients present the signs of marked inspiratory dyspnoea, but particularly of the most intense expiratory dyspnoea. The auxiliary muscles of respiration are employed in inspiration, but particularly in expiration. The abdominal muscles, especially the *recti abdominis*, are among the muscles employed in expiration. The lower ribs are drawn inwards by the marked contraction of the *transversi abdominis*.

Inspiration is slow, but expiration is prolonged to a much greater extent, being two or three times as long as inspiration.

The apex beat of the heart may disappear during the attack in pa-

tients in whom it is visible in the intervals between the attacks (projection of the dilated left lung in front of the heart).

Many patients maintain a forced (passive) position of the body. According to the degree of dyspnoea, the patient assumes an elevated dorsal or sitting position, or is bent over forwards.

The features generally express unspeakable anguish. The signs of increasing cyanosis appear upon the skin and mucous membranes. The cervical veins dilate to the thickness of a finger, the eyes become more prominent, and subconjunctival hemorrhages may occur. The latter symptoms are the evidences of excessive venous stasis.

If the attack lasts a long time, the skin may be covered with a cold, clammy sweat. The patient may suffer from a confused feeling in the head, tinnitus aurium, and flashes of light. The face grows pale and livid. In very severe cases, consciousness becomes clouded, and delirium and muscular twitchings may supervene. These symptoms are the result of excessive venous congestion of the brain and carbonic acid poisoning.

On palpation, the wheezing, sibilant rhonchi may be felt as bronchial fremitus. From theoretical considerations, we would expect the vocal fremitus to be diminished during the attack. In many cases we can convince ourselves by palpation that the diaphragm is contracting normally during the attack.

The skin usually feels very cool, and the thermometer does not show any elevation of the bodily temperature. The pulse is usually rapid, small, and very tense.

Dulness on percussion is absent in uncomplicated bronchial asthma. Dulness may be looked for only when atelectasis has developed over large parts of the lungs. The percussion note is often remarkably loud and deep, and has a tympanitic quality, particularly over the lateral and posterior regions of the thorax. Biermer attributes the production of this sound to increased tension of the alveolar tissue.

It is very characteristic of bronchial asthma that the inferior and anterior median borders of the lungs are pushed downwards and towards the sternum, and they move very little or not at all during respiration. These changes are the result of acute dilatation of the lungs. The upper border of the liver is one to three intercostal spaces lower than normal, and the area of cardiac dulness is very considerably diminished. The boundaries of the area of dulness are almost unchanged during inspiration and expiration. After the cessation of the attack, the hepatic dulness returns to its normal position, and the area of cardiac dulness resumes its former dimensions. These changes may be completed in the course of a few minutes. If the attacks occur often, the change in position of the borders of the lungs remains permanent, on account of the development of pulmonary emphysema.

The loud wheezing and whistling sounds are either absent during inspiration, or much less marked than during expiration. They are the result of stenosis, and we may therefore conclude that the bronchial stenosis is especially intense during expiration.

As the attack approaches its termination, the sibilant râles diminish, and are replaced by fine and medium moist râles. At the same time, a scanty muco-purulent sputum is expectorated.

As a rule, the vesicular respiratory murmur is not heard during the asthmatic attack, not alone because it is concealed by the loud sibilant râles, but also because it is weakened or extinguished by the narrowing

FIG. 57.



Asthma spirals. After Cursehman. Enlarged 70 times. *a*, with fibrillated ends; *b*, with delicate transverse folds; *c* and *d*, with central threads (inclosed in mucus).



of the bronchi. Preceded by a hissing sound, the vesicular murmur is sometimes heard momentarily in certain places—the impression being created as if a narrowed spot had been suddenly dilated by the respiratory current of air.

The patient's voice is generally hoarse and low. Many patients speak in a falsetto tone. At the same time, speech is often interrupted by the irregular respiratory movements.

During the attack, the heart sounds are often extremely feeble, because the distended median borders of the lungs pass in front of the heart.

Expectoration is generally absent until the close of the attack, when one or more tablespoonfuls of a grayish-white, vitreous, sticky, frothy sputum are expectorated. This is sometimes as thick and tough as gelatin. In addition to various flocculi and threads, the sputum always

FIG. 58.



Leyden's asthma crystals (Charcot-Neumann's crystals). Enlarged 500 times.

contains grayish-yellow, grayish-white, or yellowish threads, known as spiral threads or spirals. They attain a diameter of 0.5–1 mm., a length of 2–3 cm. or even more (10 cm. in a case reported by Curschmann). According to Curschmann, these threads are formed in the finest bronchi.

On microscopical examination, these spirals, which are very firm and tough, are found to be composed of thread-like structures, which are often wound around one another (Fig. 57, *b* and *c*), but occasionally are separated at the ends (Fig. 57, *a*). They often contain a bright central thread (Fig. 57, *d*) and may be inclosed in mucus (Fig. 57, *c* and *d*). Nests of round cells are found in the surrounding masses of mucus, and also within the spirals. In the latter they are found particularly in those places which present an opaque yellowish or yellowish-green appearance. Upon compressing these places with the cover-glass, we often notice an earthy resistance and a grating sound. Many round cells have

undergone granular degeneration, and constantly contain peculiar pointed crystals (Leyden's asthma crystals). These crystals are double pyramids, and, on pressure with the cover-glass, not infrequently split into transverse pieces (Fig. 58).

They vary in size, but are usually smaller at the beginning of the seizure. Towards the end of the attack they grow longer, but often present a corroded appearance. Their shape and reaction are the same as those of the Charcot-Neumann crystals obtained from the bodies of leukæmic individuals (soluble in warm water, ammonia, liquor potassæ and sodæ, acetic, hydrochloric, nitric, and sulphuric acids; insoluble in cold water, ether, alcohol, and chloroform; in glycerin they swell up until they become invisible). Ungar produced the crystals artificially, by keeping the sputum in the moist chamber, and it was then shown that they are connected locally and genetically with the round cells. Friedreich and Huber believe that they are composed of tyrosin; according to Salkowsky they are a mucin-like substance; Scheiner believes that they are composed of phosphoric acid associated with an organic base.

These spirals were found by Escherich in fibrinous bronchitis, by Curschmann in bronchial catarrh, and by Vierordt and Jaksch in croupous pneumonia. In asthma, however, they occur not alone in exceptional cases, but are present almost constantly.

Leyden's asthma crystals have also been seen in the blood and many organs of those who have died of leukæmia; also in bronchial catarrh, in the cases of fibrinous bronchitis, in phthisis, in the exudation of empyema, and in the prostatic secretion.

In one case Ungar observed crystals of oxalate of lime during the asthmatic attack, in addition to Leyden's crystals. The patient was not suffering from oxaluria.

If the sputum of asthmatic patients is exposed to the air for some time, it assumes occasionally a green color. This is the effect of a green pigment which may be extracted with alcohol.

The clinical symptoms can be best explained on the theory that the attack is the result of a spasmodic contraction of the smooth muscular fibres of the bronchi. According to Rindfleisch, the finest bronchial ramifications are surrounded by a sort of muscular sphincter. The stenosis of the finer bronchi necessarily disturbs inspiration as well as expiration, but more particularly the latter, because the action of the bronchial spasm is still further increased by the expiratory pressure to which the fine terminations of the bronchi are also subjected. These mechanical disturbances are apt to produce acute dilatation of the lungs because the normal amount of air cannot leave the alveoli during expiration, while a certain amount of air enters during inspiration. The prompt therapeutic effect of large doses of narcotics also favors this theory. Th. Weber attributes the asthmatic attack to vaso-motor disturbances. Wintrich and others regard it as the result of a tonic spasm of the diaphragm. This theory is disproven by the fact that we can often convince ourselves of the action of the diaphragm during the seizure.

Some authors maintain that the asthmatic attack is nothing more than a catarrhus acutissimus, associated with very considerable swelling of the bronchial mucous membrane. It may be said in opposition to this view that catarrhal symptoms are not infrequently absent at the beginning of an asthmatic attack.

In view of the discovery of the spirals and asthma crystals, the most

plausible theory seems to be that there is an asthmatic broncho-catarrh, which, if sufficiently extensive, gives rise to reflex spasm of the bronchial muscular fibres.

With regard to the asthmatic disposition, we need only add that in certain cases there is a serious disturbance of the entire nervous system, as is evident from the fact that asthma may alternate with other neuroses. Thus, asthma has been known to alternate with epileptic attacks, hemiparesis, or angina pectoris.

IV. DIAGNOSIS.—The diagnosis is easy in the majority of cases.

In paralysis of the crico-arytenoidei postici, particularly in hysterical individual, attacks of dyspnoea also occur, but they are always of the inspiratory type. This is also true of the dyspnoea of croup and stenosis of the larynx or trachea by foreign bodies or compression from the outside. In the rare forms of clonic spasm of the diaphragm, the thorax remains for some seconds in the inspiratory position, and inspiration is short or spasmodic as in singultus. At the same time the epigastrium becomes prominent, and the heart moves downwards and towards the median line. The spasmodically contracted diaphragm can be felt by the hand. The patient often complains of pain in the diaphragmatic region.

The differentiation from ordinary dyspnoea is usually easy; the onset and course of the dyspnoea hardly allow any diagnostic doubt to arise.

If the asthmatic attacks occur only during sleep, we may be misled by the statements of the patient to mistake bronchial asthma for nightmare. The latter, however, disappears as soon as the patient awakes, while asthmatic attacks then begin.

V. PROGNOSIS.—As a general thing, the prognosis is not unfavorable. When death from carbonic-acid poisoning threatens, paralysis of the bronchial muscles almost always occurs and the spasm ceases. Death during an attack of asthma has rarely been observed.

The prognosis as regards permanent recovery depends in great part on the nature of the primary disease. If the latter can be relieved, the asthma disappears permanently in many cases.

Asthma which develops during childhood is more often susceptible of complete recovery than that which is acquired in later years. Not infrequently, however, the disease continues for life.

VI. TREATMENT.—During an asthmatic seizure the patient should be relieved of tight articles of clothing, and kept in a large, well-ventilated apartment. Some patients experience relief from certain household remedies, such as strong coffee, swallowing pieces of ice, smoking a cigar, etc. Certain extrinsic circumstances sometimes exert a very favorable effect. Thus Trousseau describes a case in a gentleman, whose attacks were very much relieved as soon as the light in his room was made brighter.

Among drugs, narcotics possess the most certain effect. Chloral hydrate is especially useful (2.0 taken in a glassful of sugar water and repeated in half an hour if necessary). A full dose is more certain in its effect than smaller repeated doses. Hypodermic injections of morphine (morphin. hydrochloric., 1.0; glycerin, aq. distil., aa 15.0, one-half syringeful subcutaneously), morphine in powder (0.015), and other preparations of opium have been recommended, but their effects do not surpass those of chloral.

Penzoldt and others have recently claimed successful results from the use of extract quebracho.



Inhalations (chloroform, amyl nitrite, ether, ammonia, iodide of ethyl, etc.) have been recommended, but we have not recognized their superiority to chloral hydrate. This class of remedies also includes the burning of arsenic or saltpetre paper, the smoking of stramonium, hemp, arsenic or camphor cigars. The cigarettes d'Espie, which have been much employed, consist of belladonna, hyoscyamus, stramonium, and opium. Gérard's cigarettes contain belladonna, stramonium, saltpetre, and ground poppy capsules. Bombelon recommended cigarettes which were impregnated with the alcoholic extract of the leaves and tips of the twigs of *grindelia robusta*.

If the stomach is spoiled or overfilled, the administration of an emetic (apomorphin 0.1 : 10, one-half syringe-ful subcutaneously) may be very effective.

In order to prevent the recurrence of the attacks, special attention must be paid to the primary cause. This may require various modes of treatment (surgical, gynecological measures, internal medication). This indication is met with the greatest difficulty in essential bronchial asthma.

If the asthma is the result of obstinate bronchial catarrh, we may employ compressed or rarefied air, but we must be prepared to find that the first attempts at inhalation give rise to attacks of asthma. Inhalations of oxygen have been extolled. In many cases great good is effected by a change of residence.

In gouty and dyspeptic cases, we should regulate the diet strictly, and order cures in Carlsbad, Kissingen, Marienbad, Homburg, Tarasp.

If the attacks are periodical, the administration of quinine may prove very serviceable.

In nervous individuals, resort should be had to the nervines: bromide of potassium, valerian, arsenic, auronatrium chloratum, zinc, copper, and silver preparations, etc.

In many cases, the prolonged administration of iodide of potassium (10 : 200, one tablespoonful t. i. d.) is attended with benefit. We should always begin with this remedy if there are no other special indications.

Leyden recommends inhalations of common salt and carbonate of soda (ãã 1.0 : 100) several times a day, in order to dissolve the asthma crystals and thus prevent a repetition of the attack.

Schmitz obtained good results from the use of galvanism, the electrodes being placed alongside the thyroid cartilage. Neffel recommends the application of one pole (preferably the anode) to the cervical pneumogastric, while Brenner places the anode on the back of the neck, the cathode on the pneumogastric between the larynx and the sterno-mastoid. Caspari applies the cathode to the sacrum, the anode labile along the entire spine (ten to twenty minutes daily). Schaffer employed a strong faradic current, both electrodes being placed below the angles of the lower jaw, or at the level of the thyroid cartilage.

#### 4. Dilatation of the Bronchi. *Bronchiectasia*.

I. ANATOMICAL CHANGES. — Bronchial dilatation may be cylindrical, spindle-shaped, or sacculated. As a rule, the two latter are secondary to the first. Bronchiectasis occurs most frequently in the medium-sized bronchi, more rarely in the smaller ones, and most rarely in the large bronchi. They occur with equal frequency in one or both lungs, but are more frequent in the lower and middle lobes. Sometimes only a

few bronchi are affected, sometimes the larger part of the bronchial tree. Isolated circumscribed dilatations are very rare.

Cylindrical dilatation is easily recognized. If the bronchi of medium calibre are followed peripherally, they are found to retain the same lumen, or even appear dilated, instead of growing smaller. As a rule, they end in a sort of knob. If the dilatation extends to the periphery of the lung, the parenchyma is found on section to contain large cavities.

Spindle-shaped bronchiectasis is a variety of the cylindrical form, and is almost always associated with it. Several spindle-shaped dilatations sometimes follow one another in the same bronchus (rosary-shaped bronchiectasis). The intervening portions are not infrequently unusually narrow, and, in fact, the association of dilatation and stenosis of the bronchi occurs very frequently. According to some writers, rosary-shaped bronchiectasis is especially frequent in children after whooping-cough.

Sacculated bronchiectasis is a sudden and abrupt dilatation of the lumen. It may be partial or total, according as it affects only one side or the whole of the lumen of the bronchus. It is rare to find only one sacculated dilatation. They sometimes follow one another like a rosary, or several sacs may be situated alongside of one another. In the latter event, the intervening parenchyma usually presents great changes. It is generally converted into a non-aërated, fibrous tissue, which, on section, possesses a cicatrix-like, sometimes almost cartilaginous consistence, and is either very black or blackish-gray, sometimes snow-white in color (interstitial pneumonia).

The size of the sac may vary from that of a pea to that of a hen's egg or even larger. In rare cases, a single dilatation may occupy the larger part of one lobe. The bronchi passing into the sac from the trachea often present stenosis or spindle-shaped or cylindrical dilatation. The bronchi which pass from the cavity to the periphery of the lung are almost always obliterated and occluded, so that the sacculated bronchiectasis forms a sort of blind passage. Gradual obliteration of the bronchi leading into the sac also occurs at times, and the bronchiectasis is thus converted into a closed, cyst-like cavity, filled with fluid. The fluid is at first purulent, but may finally undergo cheesy or calcareous degeneration (so-called pulmonary calculi). In other cases the fluid becomes serous in character.

The mucous membrane of the dilated bronchus is almost always changed. In some cases it is very loose and thrown into folds. The mucosa and submucosa sometimes present inflammatory hyperplastic thickenings, the results of chronic inflammation. The mucous membrane may be covered with small excrescences, like villi. In the sacculated dilatations, the mucous membrane is not infrequently extremely thin, and occasionally pale, so that it looks like a serous membrane.

In not a few cases, the surface of the mucous membrane in the bronchiectasis presents a peculiar net-work appearance, with more or less prominent longitudinal and transverse ridges (trabecular degeneration). This is the result of the disappearance, in places, of the smooth muscular fibres and elastic fibres, and increase of the fibrous tissue of the mucous membrane.

Losses of substance are observed occasionally in the mucous membrane. This occurs most frequently when the secretion produced in the dilatation undergoes putrid decomposition. These losses of substance may give rise to erosion of the vessels and bronchial hemorrhage, or to perfo-

ration of the wall of the bronchi and pulmonary gangrene. If sacculated bronchiectases are situated alongside one another, they may thus be brought into abnormal communication. In rare cases, miliary and sub-miliary tubercles are found on the mucous membrane of the dilatation.

In large bronchiectases, the ciliated epithelium may be converted into mucous cells or pavement epithelium. The mouths of the mucous glands are often dilated into a funnel shape. Trojanowsky claimed that atrophy of the smooth muscular fibres occurs in all cases, but this has been disproven. The bronchial cartilage may also be implicated in these changes. Fitz described enlargement of the cartilage cavities, proliferation of the cartilage cells, compression atrophy of the basement substance, and thus a conversion of the cartilage into a sort of granulation tissue. Calcification of the cartilage is rare. Recovery of bronchiectasis sometimes occurs. Fibrous adhesions form within the dilated tube, and by their retraction cause the walls to approach one another more and more; this may finally produce obliteration of the bronchus.

Numerous changes are found in other organs. The non-dilated bronchi occasionally present evidences of catarrh. The lungs very often present proliferation of the interstitial connective tissue and retraction. In other cases, there is catarrhal or cheesy inflammation, occasionally fresh pneumonic processes. We often find emphysema, either circumscribed or involving an entire lung. Partial or total adhesions of the pleuræ are often observed.

The right heart is often dilated and hypertrophied, the muscular tissue is occasionally fatty. The other organs often present the appearances of stasis.

It must be remembered that we have referred only to chronic (persistent) bronchiectasis, not to the acute (transitory) form. In acute bronchitis, pertussis, and hypostasis of the lungs, acute dilatation of the bronchi may occur, and disappear as soon as the primary affection has subsided. If the primary disease lasts for a long time, acute bronchiectasis may become chronic.

II. ETIOLOGY.—Bronchiectasis is always secondary to chronic affections of the bronchi, pulmonary parenchyma, or pleura. It is observed after chronic bronchial catarrh, particularly bronchiolitis. In bronchial stenosis, for example, as the result of foreign bodies, bronchial dilatation develops not infrequently below the site of stenosis.

The pulmonary diseases which may give rise to bronchiectasis are: catarrhal, cheesy, and interstitial inflammation, hypostasis, retraction, emphysema.

Inflammations of the pleura often give rise to bronchiectasis, as soon as they have resulted in total or partial adhesions of both surfaces of the pleura.

Bronchiectasis is most frequent at an advanced age; it is more common in men than in women. Its development is favored by a feeble constitution; it is more common among the lower classes. According to Grainger Stewart, heredity exerts some influence.

Congenital bronchiectasis has been observed in a few instances. The dilated bronchi sometimes formed cystic spaces, filled with serous contents, and were so closely aggregated as to be almost in contact. The pulmonary alveoli were sometimes very much dilated in places.

In determining the forces which give rise to dilatation of the bronchial lumen, we must take into consideration the abnormal conditions of pressure of the respired air. These may exist during inspiration or ex-



piration or during both phases. If occlusion of the small bronchi has developed during bronchiolitis, or the alveoli have become impermeable to air on account of inflammation and hypostasis, the pressure of the inspiratory current of air will be exerted to an increased extent upon the bronchi which have remained patent. Moreover, inspiration is attended usually with the exertion of a greater amount of force than normally. Pleuritic adhesions act in the same manner. At the site of the adhesion the lung must follow the thoracic movements directly. The pressure of the inspiratory current of air is therefore distributed unequally over the bronchial distribution, and thus presents conditions for the development of bronchiectasis. All coughing spells may act injuriously by producing abnormal elevation of pressure of the air during expiration. Such conditions are noticed particularly in broncho-stenoses; for example, those produced by foreign bodies.

When bronchiectasis is the direct effect of diseases of the bronchi, we have to deal with changes in their walls. The inflamed bronchial wall becomes less capable of resistance.

Changes in the pulmonary parenchyma are important in many cases of bronchiectasis. Corrigan first showed that interstitial fibrous proliferation with subsequent retraction of the pulmonary tissue may produce bronchial dilatation in a mechanical manner, because the bronchial walls, which are implicated in the connective tissue proliferation, become separated from one another. But it is not justifiable to regard the sacculated shape of these dilatations as an undoubted evidence of this mode of development.

The softening of cheesy masses may favor the dilatation of bronchi situated within them, partly because they deprive the bronchi of supporting tissue, partly because they directly involve the walls of the bronchi.

III. SYMPTOMS.—Bronchiectasis remains entirely latent in many cases, because it produces no symptoms beyond those of chronic bronchial catarrh. This is particularly so when the dilatation, although extensive as regards length, is not very great on transverse section, and the secretion in it is not excessive.

In other cases, bronchiectasis may be suspected from the mode of expectoration and the character of the sputum. The patients do not cough often, but expectorate extremely large amounts at a time. The expectoration occurs in jets or streams, bursts from the mouth and nose, and not infrequently causes vomiting. The patients often maintain a certain position of the body, because a change gives rise at once to cough and expectoration. The greatest amount is voided in the morning after the secretion has accumulated in the dilated tubes during the night.

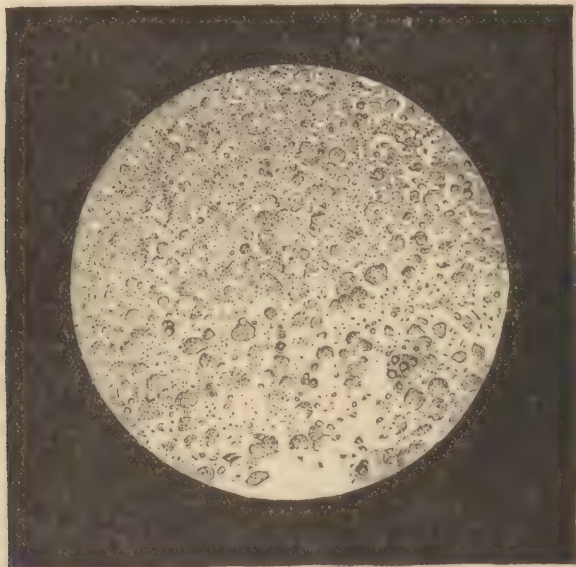
In many cases the sputum is purulent, and has a peculiar sour odor, like that of sweat. If it stands for a little while, it separates into three (more correctly into four) strata, the lower being granular and sediment-like, the middle one thin and serous, the upper one mainly frothy. Beneath the upper one is a layer of little balls of pus. The amount expectorated in twenty-four hours may greatly exceed 1,000 ccm., so that there is a surprising contrast between the physical signs and the large amount of sputum.

The sputum is composed mainly of round cells, which are partly swollen, partly fatty or granular (Fig. 59). Needles of the fatty acid are also present, either singly or in groups. Elastic fibres are also found, if the wall of the dilated bronchus is ulcerated. We sometimes

find red blood-globules, and these, if present in large numbers, may impart a clay color to the sputum. In very rare cases, the sputum contains hæmatoidin crystals. Bierman found pigmented shreds of connective tissue in several cases.

If latent bronchiectasis has existed for a certain length of time, decomposition of the sputum occurs not infrequently, and it assumes appearances which have been described as characteristic of putrid bronchitis (*vide* page 217). We again call attention to the fact that the putrid sputum is not a positive indication of bronchiectasis, since it also

FIG. 59.



From the sediment layer of the sputum in bronchiectasis. Degenerated and fatty round cells.  
Enlarged 275 times.

occurs in simple chronic bronchitis. The extension of the putrid process to the pulmonary parenchyma and the development of gangrene of the lungs are recognized by the appearance of shreds of lung tissue in the sputum.

In certain cases of bronchiectasis, a nummular or globular sputum is expectorated. These forms are also found in cases of phthisical cavities. In the globular sputum of bronchiectasis, the balls have a villous surface, sink in water, and contain an abundant granular detritus.

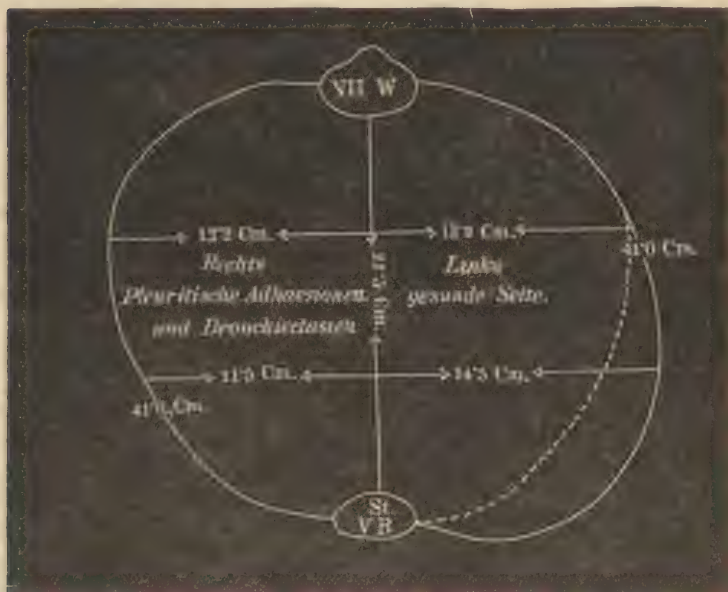
The diagnosis of bronchiectasis may be made positively if, in addition to the symptoms mentioned, local changes which indicate the existence of cavities can be demonstrated in the air passages.

On inspection of the thorax, we occasionally find flattening of the posterior and lateral portions. This is the result, in the main, of the fibrous processes of retraction in the lungs, in the vicinity of the dilated bronchi. If the bronchiectasis has followed extensive pleurisy, the

whole side may be retracted, though most markedly over the site of bronchiectasis.

The respiratory movements are diminished over these parts, and inspiratory retraction may also be noticed. The patients often assume certain passive positions. In this manner, the secretion is allowed to remain as long as possible in the bronchiectasis, and only rises gradually until it reaches the sensitive mucous membrane, and then produces cough. The position of the patient, therefore, depends on the point of entrance of the bronchus into the dilatation, the object being to prevent as long as possible the flow of secretion into the bronchus. The patients usually lie upon the affected side, more rarely on the healthy side; very rarely they assume a sitting position. The conditions are especially unfavorable in bronchiectasis of the upper lobes, when the bronchus enters from below.

FIG. 60.



Cyrto-meter curve of the thorax in bronchiectasis of the right side in a man æt. 57 years. The dotted line shows the difference between the two sides of the thorax. VII. W., 7th dorsal vertebra. St. V. R., sternal insertion of 5th rib.  $\frac{1}{2}$  natural size.

The symptoms on palpation vary from time to time in the same patient. If large bronchiectases, surrounded by non-aërated lung, are situated immediately beneath the pleura, the vocal fremitus is increased if the bronchiectasis contains no secretion, but absent if it is filled with fluid.

If the thoracic curve is taken with the cyrtometer, the flattening of the chest is readily seen graphically (Fig. 60).

Percussion reveals cavernous signs if the bronchiectasis is situated sufficiently near the surface to be affected by the vibrations produced on percussion. The deeper the cavities are situated, the more vigorously must percussion be performed. The tympanitic note is characteristic of



cavities, and this has a metallic quality if the cavity has a diameter of 6 cm. (rarely less), and has smooth walls. The percussion sound is dull tympanitic if the dilatation is surrounded by non-aërated lung. But the phenomena described are observed only when the cavity contains air. If it is filled with secretion, the tympanitic sound disappears and simple dulness takes its place. The variation in the percussion note, together with the varying intensity of vocal fremitus, is useful in diagnosis.

If the bronchus leading to the dilatation is open, cracked-pot resonance is produced; the percussion note becomes higher in pitch on opening the mouth, lower in pitch on closing the mouth.

Bronchial breathing, sometimes with a metallic quality, is heard on auscultation over large, superficial bronchiectases. But if the bronchiectasis is situated deep and covered by aërated lung, the bronchial breathing is concealed by the vesicular respiratory murmur of the aërated portions of lung. Under all circumstances, however, the bronchial breathing is absent if the cavity is filled with fluid. The presence of fluid in bronchiectases is shown by the occurrence of moist râles. These are often coarse, but usually mixed with fine râles. If the bronchiectasis is situated near the periphery, the râles have a clear sound. If the surrounding parts are non-aërated, they acquire consonance, and if the cavities are sufficiently large and smooth-walled, they assume a metallic quality. The consonance disappears if the bronchiectasis is situated deeply and is surrounded by aërated lung. But since, under these circumstances, all phenomena on palpation and percussion are absent, the constant presence of râles at a certain part of the thorax may be very important in diagnosis.

Extremely loud râles are heard occasionally in bronchiectasis, which are sometimes produced by the systole of the heart; they may be audible at some distance from the patient.

The bronchophony is increased over superficial cavities, and associated with a metallic quality if the well-known physical conditions are present. If the dilatation is filled with fluid, bronchophony is abolished.

Persons suffering from bronchiectasis may enjoy comparatively good health for a long time (twenty years or more). The general appearance and strength may be very little impaired despite the great losses entailed by the copiousness of the sputum. Marked constitutional symptoms are absent not infrequently, even when the secretion has become putrid.

In other cases, febrile movement is observed, probably as the result of absorption of the bronchial secretion. Chills occur, the fever assumes a hectic type, the patients emaciate more and more, diarrhœa develops, followed by œdema of the skin, and finally by death from exhaustion.

In some cases death is the result of cardiac disturbances. Dilatation and hypertrophy of the right heart result from the increased pressure in the pulmonary artery which has followed the changes in the lungs. If the vigor of the right heart diminishes, symptoms of stasis will be produced and may result in death.

In a third series of cases, death is the result of complications or sequelæ. Among the complications we will first mention hæmoptysis. A slight admixture of blood in the sputum is often observed as the result of violent cough. Extensive hemorrhage is rarely produced in this manner. In some cases the hemorrhage occurs spontaneously and is then the result of erosion of a large vessel. It may produce suffocation by suddenly filling the air passages.

One of the most serious complications is gangrene of the lungs, following putrid bronchitis. The fatal termination occurs more rapidly in such cases than in uncomplicated putrid bronchitis.

In very rare cases, the bronchiectases project externally like a hernia and threaten perforation, or a peripheral bronchiectasis bursts and gives rise to pyo-pneumothorax. In two cases, Mensel observed suppuration of the bronchial lymphatic glands, which gave rise to rupture into the pulmonary artery, and in the second case also to rupture into the œsophagus and into a bronchus.

During the course of bronchiectases, inflammations of the joints sometimes occur. They are similar to the joint affection of acute articular rheumatism, and are probably the result of absorption of bronchial secretion and the deposit of inflammation-producers in the joints.

Amyloid degeneration of the abdominal organs occurs not infrequently as a sequel. Cerebral abscesses (probably of metastatic origin) have also been found in bronchiectasis. Nothnagel recently described an abscess of the spinal cord. Barth observed meningitis and meningo-encephalitis.

Death is not infrequently the result of intercurrent pneumonia.

IV. DIAGNOSIS.—As a rule, the recognition of bronchiectasis is easy if the characteristic sputum and mode of expectoration and the signs of cavities are present in combination.

In distinguishing bronchiectasis from phthisical cavities, we must take into consideration the hereditary conditions and the development of the disease. The following signs are indicative of phthisis: the phthisical appearance, situation of the cavities near both apices, scantier sputum, smoother surface of the balls in the "globular" sputum, larger number of elastic fibres in the sputum, infrequency of a rapid change in the physical signs, absence of the "mouthful" expectoration.

The development of the disease is an important factor in the differential diagnosis from sacculated pyo-pneumothorax which has ruptured into the lung. In addition, the secretion of the empyema not infrequently has a strong smell of sulphuretted hydrogen. Finally, crystals of cholesterin and hæmatoidin are found in the pus of empyema, while cholesterin is never, and hæmatoidin is rarely found in the secretion of bronchiectasis.

Putrid bronchitis within bronchial dilatations is distinguished from pulmonary gangrene by the presence, in the latter, of shreds of pulmonary parenchyma in the sputum.

In cases in which we must depend solely on the expectoration and sputum in making a diagnosis, mistakes cannot always be avoided. The following possibilities must be considered.

a. The case may be regarded as simple bronchitis (this cannot be avoided in cases of scanty secretion in bronchiectases).

b. Putrid bronchitis in simple chronic bronchitis may be mistaken for that occurring in bronchiectasis.

c. It may be regarded as abscess of the lung if the sputum is purulent. It should be remembered that shreds of lung tissue, cholesterin crystals, and hematoidin are found in the sputum of pulmonary abscess.

V. PROGNOSIS.—The recovery of bronchiectases, if it occurs at all, is so exceptional that it need not be taken into consideration. The prognosis is also unfavorable for the further reason that during the course of the disease a series of accidents may occur which cannot be prevented, and often result fatally with surprising rapidity.

VI. TREATMENT.—The discharge of the secretion is best effected by

means of expectorants or, in certain cases, of emetics. Among the former we may particularly recommend ipecac, senega, liq. ammon. anisat., benzoic acid (vide page 220). Gerhardt saw good effects from compression of the thorax during expiration at certain intervals, but this procedure may be followed by hemorrhage from the air-passages and muscular twitchings.

The diminution and disinfection of the secretion are best effected by means of the balsams, particularly the inhalation of turpentine or carbolic acid. The use of Curschmann's mask (vide article on pulmonary gangrene) deserves special recommendation. Very little can be expected from the use of astringents (for example, acetate of lead, tannic acid, alum, liq. ferri sesquichlorati). To secure disinfection alone, we may employ—either internally or by inhalation—the various antiseptics, such as carbolic acid, salicin preparations, creasote, thymol, resorcin, etc.

The diet should be nourishing, and stimulants should be given in sufficient quantities.

Surgical measures have been adopted recently in the treatment of bronchiectasis. Seiffert injected 2.0 of a two-per-cent solution of carbolic acid directly into the lungs at the affected spot. After each injection, the quantity and smell of the sputum were lessened. In one case, Marshall opened and drained the superficial bronchiectasis; the amount of sputum was diminished, but the patient died of cerebral abscess. Lauenstein employed drainage successfully in a case of bronchiectasis of the right upper lobe.

The chief difficulty in the way of operation is our inability to determine positively the depth, size, and number of the cavities in the lung.

### 5. *Narrowing of the Bronchi. Bronchostenosis.*

I. ETIOLOGY.—Stenosis of the bronchi may be produced by changes in the lumen, in the wall of the bronchus or outside of it.

Intrabronchial stenoses are most frequent, and are the result of the presence of mucus, pus, blood, fibrinous exudation, or foreign bodies within the tubes. These conditions will not be discussed at the present time. Infrabronchial stenoses form the most infrequent variety. They are the result of cicatricial strictures, generally of a syphilitic character. The stenosis extends, as a rule, to the trachea and even to the larynx, and is most marked in these organs. Demarquay reported a unique case in which a glanders ulcer had given rise to bronchostenosis. I observed bronchostenosis in three cases, as the result of rupture of suppurating bronchial glands into the air-passages. In very rare cases, inflammatory hyperplasia of the bronchial mucous membrane gives rise to bronchostenosis. This may also be produced by tumors which have either developed in the wall of the bronchus, or have proliferated into its lumen from surrounding parts.

Extrabronchial stenoses are always the result of compression.

Tumors of the thyroid gland may cause stenosis of a main bronchus, but more frequently they compress the trachea. It is also said that enlargement of the thymus may produce the same effect. A very frequent cause is enlargement of the tracheal, bronchial, and mediastinal lymphatic glands, whether the result of scrofula, tuberculosis, syphilis, or neoplasms. The enlarged glands sometimes give rise to compression within the lungs. Bronchostenosis may also be the result of inflammatory and other processes in the mediastinum. Aneurism of the aorta or innomi-



nate artery often gives rise to bronchostenosis, particularly of the right main bronchus. This is also true of diseases of the pericardium. According to King, a markedly dilated left auricle may compress the left main bronchus. In rare cases, bronchostenosis is the result of compression by tumors of the œsophagus or lungs.

II. ANATOMICAL CHANGES.—The anatomical changes depend upon the cause of the affection. If the stenosis is caused by a foreign body, the latter will be found either firmly wedged in or freely movable. Cicatricial strictures appear as transverse septa projecting into the lumen of the bronchus, or the stricture runs along the length of the bronchus. This is also true of the stenoses resulting from hyperplasia of the mucous membrane. Tumors generally appear as projecting fungoid proliferations. In compression-stenosis, the lumen of the bronchus either becomes triangular or assumes a slit shape.

The bronchus is very often dilated below the site of the stricture, and the mucous membrane may be inflamed. The parenchyma of the lungs is often in a condition of collapse, emphysema, inflammation, gangrene, or abscess.

III. SYMPTOMS.—The clinical history is most distinct when foreign bodies suddenly occlude a main bronchus or a large trunk. The symptoms then occur suddenly, as a rule; when due to other causes, they generally develop gradually, and are often accompanied by the symptoms of the primary disease.

If the main bronchus is occluded, the corresponding side of the thorax takes less part in the respiratory movements than the healthy side. At the same time the signs of objective dyspnoea are noticed. The difficulty in respiration chiefly affects inspiration, and the inspiratory auxiliary muscles are brought into play. Inspiration is prolonged, and the rapidity of respiration is generally diminished. The yielding portions of the thorax (intercostal spaces, jugular and supra-clavicular fossæ, costal cartilages and ensiform processes, insertion of the diaphragm into the sternum) present inspiratory retraction, because the air in the bronchi and pulmonary parenchyma below the stenosis is excessively rarefied. The larynx and trachea usually make slight inspiratory movements downwards, but these are not as marked as in pure laryngeal stenosis. The signs of cyanosis are also noticeable.

The disappearance or diminution of vocal fremitus on palpation is a very important symptom. As a rule, bronchial fremitus can also be felt. If the bronchostenosis has lasted a long time, the circumference of the thorax on the affected side is less than on the healthy side. The vital capacity of the lungs is much diminished.

The percussion note at first is unchanged; after the stenosis has lasted some time, it becomes lower in pitch, and tympanitic on the affected side (relaxation of the lung). The lower borders of the lungs undergo very little or no change of position during respiration.

The vesicular respiratory murmur is either abolished or very feeble on the affected side. It is often concealed by loud, buzzing, or wheezing stenotic murmurs, which are sometimes so loud as to be heard at a distance from the patient. They may be heard during inspiration or during both phases of respiration, but are usually feebler, shorter, and, according to Gerhardt, higher in pitch during expiration. If the bronchus contains a movable foreign body, its movements during respiration may produce a peculiar rattling noise. The bronchophony is enfeebled or abolished.

The patients are usually tortured by a feeling of terrible fear and want of breath. They sometimes have a sensation "as if one lung is not breathing."

The unaffected lung not infrequently presents the signs of acute dilatation (compensatory).

Cough and expectoration may be absent. The more violent the cough, the greater is the bronchial dilatation which is usually found, at the autopsy, below the site of stenosis. The voice often becomes hoarse and whispering, because the vocal cords are brought in contact with a less vigorous current of air.

The pulse is sometimes slow, occasionally very rapid, particularly when the pneumogastric is compressed and paralyzed by a tumor.

If the dyspnoea is very marked, the pulse almost disappears during inspiration (vide Fig. 6, page 25). Fox also observed this phenomenon (*pulsus inspiratione intermittens*) in a case of dilatation of the right bronchus. In this case the bronchiectasis increased in size during inspiration, and compressed the aorta.

Fever may be entirely absent. Diuresis is scanty or almost entirely abolished.

In many cases there are intercurrent attacks of increased dyspnoea, the cause of which is not well known. They are occasionally the effect of purely mechanical causes, inasmuch as masses of secretion give rise to temporary complete occlusion.

If the obstruction is situated in one of the large bronchial branches, the physical signs described above are observed only in those parts which are supplied by the obstructed bronchus.

Death may occur from gradual suffocation. The patients become comatose, delirious, the cyanosis is excessive, Cheyne-Stokes respiration sometimes makes its appearance, and life becomes extinct. Sudden death from suffocation is also observed at times.

In other cases, oedema, inflammation, gangrene, or abscess of the lung are produced. Sudden death also occurs in some cases in which the cause is not revealed on autopsy. Finally, the primary disease may result in sudden or gradual death.

IV. DIAGNOSIS.—The recognition of bronchostenosis is usually easy. In exceptional cases both main bronchi are stenosed, and this condition must be differentiated from laryngeal and tracheal stenosis. This is effected, apart from the external examination of these organs, by tracheoscopic and laryngoscopic exploration.

The situation of the stenosis is generally determined by the extension of the secondary symptoms. We should also determine the locality at which the murmurs of stenosis are heard and felt with the greatest distinctness.

The character of the stenosis is determined by the examination of the adjacent organs and by the clinical history. It must be remembered, however, that foreign bodies may enter the air passages unnoticed, and give rise to symptoms of stenosis only after they have become swollen (for examples, beans, peas, etc.). The foreign body may change its position in consequence of violent coughing spells, and may even pass into another bronchus, so that the symptoms of stenosis also change their location.

V. PROGNOSIS.—The prognosis is almost always unfavorable. It depends mainly on the primary affection, the greatest chances of recovery being presented by scrofulous and syphilitic processes. The removal of

foreign bodies, which occasionally are carried around for years, often is the result of chance (for example, while laughing, coughing, etc.).

VI. TREATMENT.—In scrofulous or syphilitic cases, we may order cod-liver oil, iodine, and mercurial preparations. Good effects from the use of iodine have also been obtained in other cases.

Tumors must be removed by operative procedures. Emetics may be employed to secure the removal of foreign bodies. Attacks of dyspnoea are relieved by cautious injections of morphine. The sequelæ are treated according to the usual therapeutic rules.

## APPENDIX.

### *Diseases of the Tracheo-Bronchial Lymphatic Glands.—Adenopathia Tracheo-Bronchialis.*

1. Acute inflammatory enlargement of the tracheo-bronchial glands (lymphadenitis tracheo-bronchialis acuta), is an almost constant concomitant of acute inflammations of the air passages. The inflamed lymphatic glands are swollen to the size of a hazelnut or walnut, are very red on cross section, contain extravasations of blood here and there, and are very succulent. This condition hardly ever produces any clinical symptoms. Some French writers have attributed to this condition an important part in the production of certain diseases, for example, whooping-cough and spasm of the glottis. It is assumed that the swollen glands irritate the adjacent recurrent laryngeal nerve, and thus, by reflex action, produce spasm of the parts which produce cough and phonation.

2. Chronic lymphadenitis tracheo bronchialis is not infrequently the result of chronic inflammations of the air passages. It is sometimes left over after acute lymphadenitis, particularly in scrofulous individuals, who have a predisposition to enlargement of the lymphatic glands. The enlargement of the glands is even greater than in acute lymphadenitis. The glands have a firm feel and increased resistance; the transverse section is dry, and has a grayish-red or brownish-red color. The microscope sometimes shows a predominant increase of the connective tissue, sometimes of the cellular elements. In the former event, the gland is firm, almost tendinous; in the latter it is granular. These conditions may be associated with a series of grave symptoms.

If the glands are very large, areas of unusual dulness are observed not infrequently in the thorax. The dulness is observed most often over the manubrium sterni, and extends occasionally towards the clavicles. Or the dulness is sometimes noticed along the spine at the level of the upper dorsal vertebræ.

In certain cases, the manubrium sterni presents a distinct prominence, particularly in children in whom the bones are more yielding.

Pressure of the glands upon the recurrent nerve may cause immobility of the corresponding vocal cord. Vomiting follows compression of the vagus; difference in the pupils is the result of pressure on the sympathetic. Or symptoms of tracheal or bronchial stenosis are produced by pressure on the trachea or bronchi. Intrathoracic veins are sometimes stenosed, and this condition gives rise to dilatation of the cutaneous veins, circumscribed cyanosis and oedema.

Pressure on the aorta or pulmonary artery may give rise to systolic murmurs in these vessels. Pressure upon the œsophagus results occasionally in disturbances of deglutition.



When the glands diminish in size, they may undergo retraction, together with the surrounding connective tissue. Paralysis of the recurrent laryngeal nerve may result from the implication of the nerve in this process of retraction. Or the wall of the œsophagus may be involved, and a so-called traction diverticulum of the œsophagus is developed in consequence. Narrowing and occlusion of vessels may also be produced.

The inflamed glands sometimes undergo softening and even suppuration. This may be followed by rupture of the pus externally. If it ruptures into the trachea or one of the main bronchi, it may cause sudden death from suffocation. In other cases, the signs of bronchial blennorrhœa or putrid bronchitis develop, the real cause being overlooked. Thus, I recently made a mistaken diagnosis of abscess of the middle lobe of the lung, based upon the abundant purulent sputum and cavernous signs in this region. The autopsy showed pneumonic infiltration of the right middle lobe, and immediately adjacent a cavity, almost as large as a hen's egg, which was the result of suppuration of the bronchial glands. This abscess had ruptured into one of the right bronchi. Under such circumstances, the diagnosis can hardly be made unless the sputum contains large particles of glandular tissue. If the affected glands are calcified, the sputum may contain so-called bronchial calculi. Perforation may also occur into the œsophagus, blood-vessels, mediastinum, pleural or pericardial cavities, giving rise to embolic processes, acute mediastinitis, pleurisy, or pericarditis. Rautenberg reported a fatal case of melæna in a child. It was found to be the result of rupture of a suppurating bronchial gland into the pulmonary artery and œsophagus.

Rupture of bronchial glands is evidently not a frequent cause of death, for we often find upon the mucous membrane of the œsophagus or bronchi, or the inner surface of the pulmonary artery stellate pigmented cicatrices which could only have been produced in the manner above-described. This again offers an opportunity for the formation of traction diverticula of the œsophagus, and of stenosis of the vessels or bronchi.

3. Tubercular changes in the tracheo-bronchial glands have a very serious significance. Other glands are usually affected in a similar manner; more rarely the disease is independent. The swollen glands may produce the same pressure effects as those described above. Calcification or softening and suppuration of the glands may also occur (*vide* preceding section). Special significance attaches to the fact that these glands may form the starting-point of general or meningeal acute miliary tuberculosis.

4. Hyperplastic changes of the tracheo-bronchial glands are also observed in leukæmia and pseudo-leukæmia. Clinical symptoms, if present at all, are due to pressure on surrounding parts.

5. This is also true of tumors (sarcoma, cancer, rarely fibroma) of these glands. The neoplasms may attain such dimensions that they fill the mediastinum and pleural cavities, push back the lungs and proliferate into the pericardium.

6. Finally, the bronchial glands are very markedly implicated in conditions of pneumoconiosis, because they endeavor to retain all the particles of dust which are inhaled and enter the lymph channels of the lungs.

## PART V.

## DISEASES OF THE LUNGS.

*I. Hæmoptysis.**(Pneumorrhagia.)*

I. ETIOLOGY.—Hæmoptysis is the admixture of blood with the sputum in amounts which are visible to the naked eye. This condition is merely a symptom of other diseases.

The blood may have its origin in the larynx, trachea, bronchi, or parenchyma of the lungs. Hemorrhages from the larynx or trachea are not frequent and are observed especially in catarrh, ulcerative processes, and in aneurisms of the aorta, pulmonary, subclavian or carotid arteries. In the latter conditions the hemorrhage is generally very profuse and rapidly produces death.

In the majority of cases hæmoptysis is either a bronchial or pulmonary hemorrhage. The former is more frequent, but it may be difficult to determine positively whether the hemorrhage is bronchial or pulmonary.

The following are the causes of bronchial hemorrhage:

*a.* Inflammatory and ulcerative processes of the bronchial mucous membrane.

Hæmoptysis is sometimes seen in violent bronchial catarrh and in fibrinous bronchitis. It also occurs in the stasis catarrhs of heart disease (generally from mitral lesions, aortic stenosis, and tricuspid insufficiency, but also in affections of other valves and in diseases of the cardiac muscle). It is also observed in bronchiectasis, particularly in putrid bronchitis.

*b.* Vigorous mechanical, thermal, and chemical irritation of the bronchial mucous membrane.

Among the mechanical causes are violent cough, loud, continued talking, singing, screaming, carrying heavy loads, forced mountaineering, dancing, riding, gymnastics, and excessive bodily exertion in general. Inhalation of very cold or warm air, or irritating gases may also produce hæmoptysis. Foreign bodies in the bronchi may give rise to hemorrhage by direct injury of the mucous membrane. In other cases, the hemorrhage follows ulceration of the mucous membrane and, as a rule, it takes place as soon as the foreign body is loosened and discharged.

*c.* Pulmonary phthisis is a very frequent cause of bronchial hemorrhage. It is the result, in part, of loosening of the bronchial mucous membrane and increased friability of its vessels, in part, of fatty changes or the formation of tubercles in the walls of the vessels.

Hæmoptysis occurs not infrequently at a period when, to judge from the physical signs, the lungs are entirely healthy. It may even be repeated for several years before the first changes in the lungs

become manifest. Even at this time, however, the sputum often contains tubercle bacilli. This condition is often observed in delicate individuals of a phthisical habit, with a phthisical family history. Niemeyer believed that the bronchial hemorrhage was the cause of the phthisis which becomes manifest at a later period. This view is now discarded. It is known that the lung may be diseased, although no physical changes are demonstrable with the exception of the tubercle bacilli in the sputum, and in addition it has been shown that blood, which was injected into the trachea and bronchi of rabbits and then passed into the alveoli, is rapidly absorbed without giving rise to chronic inflammation.

*d.* Rupture of an aneurism (aorta, pulmonary artery) into a bronchus may give rise to very violent hæmoptysis.

*e.* Traumatic bronchial hemorrhage occurs very rarely.

*f.* In many cases bronchial hemorrhage is associated with general nutritive disturbances or infectious diseases.

It is observed in hæmophilia, scurvy, and morbus maculosis Werlhofii. Sturges observed hæmoptysis in a case of renal atrophy, and I have had a similar experience in cholæmia. Among acute infectious diseases we may mention the acute exanthemata, when they assume a hemorrhagic character; among chronic infectious diseases, intermittent fever deserves special mention. The injurious influence of the malarial poison is sometimes manifested solely by bronchial hemorrhage, which recurs daily at the same time, and disappears after the administration of quinine, or spontaneously after a certain length of time. In the latter event other malarial symptoms generally become more distinct.

*g.* Vicarious bronchial hemorrhage is a term applied to hæmoptysis which takes the place of hemorrhages from other organs. It seems certain that bronchial hemorrhage sometimes takes the place of the menses, but it is doubtful whether it occurs instead of previously existing hemorrhoidal hemorrhages or epistaxis.

Vicarious bronchial hemorrhage may take place when the respiratory organs are entirely healthy, but it is more apt to occur when phthisical changes are present in the lungs. Putegnat described three cases in phthisical pregnant women, in whom hæmoptysis occurred at each menstrual period.

According to Huchard, arthritis urica is a frequent cause of hæmoptysis.

The anatomical lesions which give rise to pulmonary hemorrhage are not always alike. It sometimes occurs from rupture of the blood-vessels, sometimes from diapedesis of red blood-globules (hemorrhagic infarction), sometimes not alone the blood-vessels are ruptured, but the lung tissue is also destroyed (pulmonary apoplexy).

Pulmonary hemorrhage (capillary or arterial) is most frequent in pulmonary phthisis. Arterial hemorrhages are among the late symptoms of phthisis and occur after the formation of cavities. The walls of the cavities often contain diffusely dilated or aneurismal arteries, whose rupture may produce very violent hemorrhage. The site of perforation is closed by the formation of a thrombus, but the thrombus may be suddenly loosened and removed, whereupon the hemorrhage recurs. Prior to the sixth year of life hæmoptysis is extremely rare, despite the existence of pulmonary phthisis. This is owing to the fact that cavities form very rarely in children. Moreover, the cavities forming during the phthisis of childhood usually develop centrifugally, so that the peri-



pheral vessels are generally obliterated and closed before they are drawn into the domain of the cavity.

Hæmoptysis also occurs in pulmonary gangrene and abscess, as soon as the ulcerative process affects blood-vessels in which the circulation is free. Fraentzel has recently shown that the hæmoptysis of pulmonary gangrene is usually very profuse, and occasionally is the first evidence of the disease.

Hæmoptysis is not infrequent in tumors (cancer, sarcoma) and parasites of the lungs (echinococci).

Baetz recently described a peculiar form of parasitic hæmoptysis which he observed in nineteen young Japanese, in whom the examination of the lungs gave negative results. The sputum contained psorospermia, to which Baetz applied the term *gregarina pulmonalis s. fusca*.

The sputum is found mixed with blood in acute inflammations of the pulmonary parenchyma. It is well known that the sputum is tinged with blood in the first stage of fibrinous pneumonia, but this may also occur in catarrhal inflammation.

Injury to the lung generally, though not always, leads to that form of hemorrhage known as apoplexia pulmonum. This may occur either with or without injury of the bones and soft parts of the thorax. Very extensive hemorrhages and destruction of the lungs may be produced by aneurisms which have ruptured into the lungs, often after they have grown at the expense of the pulmonary parenchyma.

Pulmonary hemorrhage occurs very often in heart disease (hemorrhagic infarction). This is not always the result of the same anatomical changes. In rare cases the capillaries are torn, in the majority of cases we have to deal with embolic or thrombotic processes. Emboli which have been carried into the pulmonary artery must have taken their origin in the right heart and are almost always derived from cardiac thrombi which generally form, after previous dilatation of the right heart, in the right auricular appendix and between the columnæ carneæ near the apex. Hemorrhagic infarctions are most frequent in mitral and tricuspid lesions because changes in the right heart are very common in these diseases. It is also observed in other valvular lesions, in idiopathic affections of the heart muscle, and in changes in the right heart following emphysema.

An embolus may also be carried through the venæ cavae into the right heart, so that peripheral venous thromboses must be looked upon as causes of hemorrhagic infarctions and pulmonary hemorrhage.

In the distribution of the superior vena cava infarctions may follow severe injury to the skull, inflammation of the petrous portion of the temporal bone, thrombosis of the cerebral sinuses, and furuncles in the face; in the distribution of the inferior vena cava, marantic thrombosis in the lower limbs, thrombosis of the ovarian and uterine veins, operations upon the hemorrhoidal veins, dysentery, thrombosis of the prostatic vein, of the renal veins. We must also refer to fatty emboli of the lungs as the result of fracture of the bones. Felz observed hemorrhagic infarction after extensive burns.

In some cases pulmonary hemorrhage is said to be of nervous origin. It has been observed in cerebral hemorrhage. Jehn often found pulmonary hemorrhage in the insane, and Carré states that it is not infrequent in chorea, epilepsy, hypochondriasis, and in cerebral and spinal diseases.

Hæmoptysis is more frequent in males than in females. It is most

common from the fifteenth to the thirty-fifth years, but is extremely rare in children and old people.

II. ANATOMICAL CHANGES.—In bronchial hemorrhage the bronchi are found to contain a greater or less amount of blood, either fluid or coagulated, fresh in appearance or blackish to brownish in color. The mucous membrane is often swollen and friable. It may be abnormally injected or reddened, in other cases it is extremely anæmic and pale on account of the very profuse hemorrhage. The bleeding point cannot be discerned, as a rule, because the hemorrhage is usually capillary in character.

As a rule, some of the blood flows into the alveoli, or is drawn into them during inspiration. It becomes difficult, therefore, to differentiate anatomically between pulmonary and bronchial hemorrhage in individual cases. The red blood-globules pass from the alveoli into the interstitial connective tissue of the lungs, and in this way the blood is rapidly absorbed.

Nothnagel found, in experiments on rabbits, that blood contained in the air passages passed into the interfundibular septa in large amounts within three and a half minutes.

For some unknown reason, absorption of the blood is checked in some cases in human beings, and the clots remain in the bronchi. They lose their color, soften, and become almost puriform in appearance. Such cases are very rare, and usually terminate in complete absorption.

In pulmonary apoplexy, greater or lesser portions of the lung are found converted into a bloody mass consisting of blood and destroyed lung tissue.

In hemorrhages from phthisical cavities the walls of the latter are often found to contain a dilated blood-vessel, in which is seen a perforation which is either open or closed by a thrombus. The cavity and its afferent bronchus are usually filled with blood or clots.

In capillary hemorrhages into the lung, the site of hemorrhage cannot, as a rule, be determined with certainty. The lung contains blood-red or blackish-red, later brownish-red spots, which are firm, non-aërated, but often surrounded by dilated and emphysematous portions of lung. The transverse section of these spots is slightly granular and, on microscopic examination, red blood-globules are found in the alveoli and interfundibular septa. The epithelium of the alveoli is not infrequently slightly swollen, granular, and partly desquamated. After a time the unabsorbed red blood-globules undergo shrinking and granular degeneration; blood pigment in the form of needles, more rarely in the form of plates, is deposited in the alveoli and interfundibular septa.

Pulmonary hemorrhage in heart disease is usually the result of embolic processes. The hemorrhagic infarctions produced in this manner are wedge-shaped, as a rule, with the base directed towards the surface of the lung, the apex towards the hilus.

Their size may vary considerably, but infarctions involving half a lobe or an entire lobe are extremely rare. The infarction is situated generally at the surface, more rarely internally. In the former event the pleura is generally inflamed over the site of the infarction.

An embolic infarction is hard, devoid of air, blackish, bluish or brownish-red, and slightly granular. On scraping it with a knife, a crumbly bloody mass is discharged. The alveolar spaces and interfundibular septa contain innumerable blood-globules.

The infarctions may undergo various secondary changes—they may dry and be absorbed, leaving a brown or black fibrous cicatrix, they may become cheesy, or undergo softening, abscess formation, and gangrene. If the pleura is involved in the destructive process, pneumothorax may develop.

The lungs may contain a number of infarctions in various stages of development, the majority being situated in the right lower lobe in the region situated between the scapula and spine. The embolus generally follows this course on account of its weight and the more vigorous current of blood flowing into the right lower lobe. If this current has been enfeebled by previous embolism, or if the circulation in the right pulmonary artery is impeded by retraction of the lung or compression as the result of pleuritic exudation, or if, when the patient is lying on the right side, the current of blood to the left is increased by the more vigorous respiratory movements of the left side, the emboli will generally be carried into the left lung.

III. SYMPTOMS.—Extravasations of blood into the air passages are found not infrequently on autopsy, although no symptoms indicative of this condition had been noticed during life. This may happen when the hemorrhages are very slight and form gradually, or when they are very extensive, and cause death by suffocation before the blood could escape externally. This is particularly true of pulmonary apoplexy.

It is rare that the hemorrhage occurs unexpectedly in the midst of apparently excellent health.

In many cases hæmoptysis is preceded by notable subjective symptoms. The patients often feel as if something warm were rising beneath the sternum and then, on coughing, blood is expectorated. This sensation is rarely felt at the source of the hemorrhage. Some patients complain of a peculiar flat, salty or blood-like taste in the mouth, the necessity of hawking is then felt, and the blood makes its appearance. Others feel merely a peculiar desire to cough, or complain of a rush of blood to the head, or of a feeling of oppression in the chest. These symptoms may precede the hemorrhage for several days.

The amount of the hemorrhage varies extremely. The blood appears not infrequently in small dots or streaks, which are mixed with the sputum. In other cases the sputum is purely bloody, in still others the blood is intimately mingled with the other constituents of the sputum. The hemorrhage may be so profuse that several pounds of blood are lost in a single day. If the hæmoptysis is very rapid and profuse, the blood sometimes bursts in a stream from the mouth and nose, and gives rise to vomiting, so that blood and articles of food are mixed together.

Sometimes a single hemorrhage alone occurs. More frequently it recurs at intervals of one-quarter to one-half hour, or a number of hemorrhages occur after the lapse of several days. In some cases they cannot be checked and death ensues.

Whether the sputum is purely bloody or merely tinged with blood, the color of the blood is almost always bright red and arterial. At the same time the purely bloody sputum is frothy and any clots which may be present also contain air bubbles. But if the blood has stagnated for some time in a large cavity, it not infrequently becomes black and contains solid clots.

The sputum which is intimately mixed with blood varies in color. It often has a peculiar clay color, particularly in putrid bronchitis and gangrene of the lungs. In hemorrhagic infarctions we often find a red-

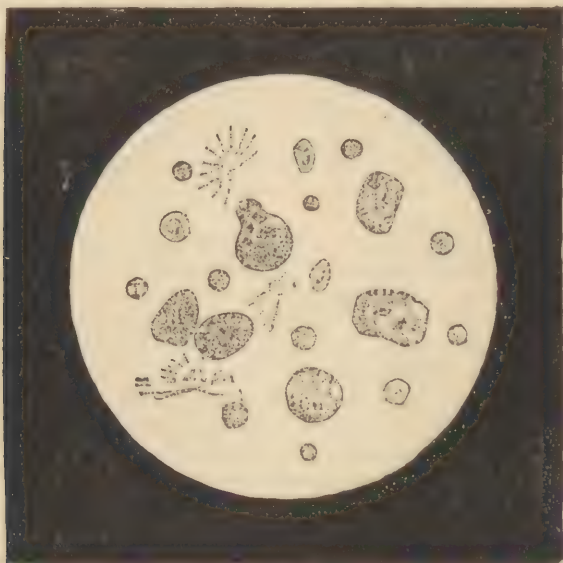


dish-brown sputum, like the rusty sputum of fibrinous pneumonia. The peculiar color is the result of changes in the blood pigment, and the appearance of a reddish-brown sputum must be regarded as the terminal stage of hemorrhagic infarction. In some cases the hemorrhage is so slight and takes place so slowly, that no fresh blood is expectorated, and the rusty brown sputum is not expectorated until some time after the formation of the infarction.

In the sputum which is tinged with blood, the red blood-globules are arranged in groups, corresponding to the dots and streaks of blood. The narrow edges of the blood-globules are in contact with one another, not the broad surfaces. In the purely bloody sputum, the blood-globules are arranged in columns.

In the bloody sputum and in that form which is intimately mixed

FIG. 61.



Sputum of hemorrhagic infarction (14th day), in a woman æt. 32 years, suffering from mitral insufficiency and stenosis. Blood pigment in granules and plates inclosed in alveolar epithelium. Enlarged 275 times.

with blood, the red blood-globules retain their normal shape for a long time, but in putrid sputa they are sometimes destroyed very rapidly. In purely bloody sputum stellate and dentate shapes are observed not infrequently. If the red blood-globules have remained in the air passages for some time, they undergo changes in form which lead finally to disintegration. They swell, become lentil-shaped or globular, assume a stellate shape, or they lose their pigment and are converted into pale, colorless disks, or they gradually undergo granular disintegration. The coloring matter which is set free may penetrate other cellular structures in the sputum, and these then assume a diffuse yellow color or are interspersed with brown granules, needles, or small plates of pigment. These changes are observed most frequently in the reddish-brown sputum of hemorrhagic infarctions (Fig. 61). On the average, a period of two

weeks elapses before the development of pigmented cells. The last traces of a hemorrhage may often be demonstrated many weeks later by microscopical examination of the sputum.

Hæmoptysis may occur without any demonstrable physical changes in the thorax. If the pulmonary alveoli are filled with blood we may hear, under favorable circumstances, fine crepitant râles which are produced during inspiration by the tearing asunder of the walls of the alveoli from their bloody contents. Dulness on percussion can only be expected if the part filled with blood is at least four to six ccm. in circumference and two cm. in thickness. Careful, gentle percussion is necessary to bring out the dulness. If a large area, which includes large bronchi, is infiltrated, we will find the physical signs of pulmonary infiltration, viz., increased vocal fremitus, dulness, bronchial breathing. But if there is not free conduction through the bronchi, vocal fremitus will be abolished and no respiratory murmur will be audible.

If the bronchi contain fluid blood, coarse and medium râles will be heard. These do not possess consonance, unless the surrounding alveoli are affected and destitute of air.

If the hemorrhage is very profuse and rapid, symptoms of suffocation may develop as the result of occlusion of large bronchi. The patients become very cyanotic, breathing is difficult, the auxiliary muscles of respiration are brought into play. During inspiration the thorax is retracted or very slightly dilated over the affected portion of the lung.

I recently observed a case in which, as the result of the violent respiratory movements and the rapidly developing vicarious dilatation of the lungs (almost the entire right lung had been rendered useless), subcutaneous emphysema occurred in the neck. This could hardly be interpreted in any other manner than as the result of interstitial pulmonary emphysema.

Special symptoms, associated with the etiology of the hæmoptysis, are sometimes observed. This is particularly true of hemorrhagic infarction. The occurrence of the embolism is manifested not infrequently by a chill; syncopal attacks may also occur. As a rule the hæmoptysis does not appear until one to three days after the occurrence of the embolism. In a case observed by Gerhardt, it appeared nine hours after the chill.

Among the complications, particular mention should be made of elevation of temperature. Gerhardt's investigations render it probable that embolism of the pulmonary artery as such may increase the bodily temperature. In other cases the fever is evidently the result of absorption of the readily decomposed and often putrid masses of blood. Inflammatory changes may also occur around infectious emboli. Finally, pleurisy—dry and fluid—is observed not infrequently in the hemorrhagic infarctions of heart disease.

Gerhardt has called attention to the occurrence of jaundice, which he regards as hæmatogenous in many cases. In very profuse hæmoptysis I have several times observed anæmic, systolic cardiac murmurs and dilatation of the right heart.

Complete recovery after hæmoptysis is not so rare as is generally believed. Hæmoptysis may prove fatal by suffocation or the loss of blood. In other cases, inflammatory changes (abscess, gangrene) occur in the lungs, or pleurisy develops and proves fatal.

IV. DIAGNOSIS.—Hæmoptysis may be mistaken for hemorrhage from the nose, pharynx, or gums.

Slight epistaxis may occur unnoticed during the night, and the blood

flow into the pharynx or into the larynx. It is expectorated by the patient on awakening, and is thus regarded as hæmoptysis. In doubtful cases, we should inquire whether epistaxis has occurred or whether the nasal secretion contained streaks of blood, and must make a careful rhinoscopic examination. Careful examination of the pharynx and gums will readily determine whether the hemorrhage comes from these structures.

Hemorrhages from the larynx or trachea may be recognized with the aid of the laryngoscope.

The differentiation between hæmoptysis and hæmatemesis may be attended with great difficulty.

*a.* In hæmoptysis the blood is discharged by coughing, in hæmatemesis by vomiting. But if the hæmoptysis is very profuse, it may give rise to vomiting and, on the other hand, the blood often flows into the entrance to the larynx during profuse hæmatemesis, and is then removed by coughing. *b.* In hæmoptysis the blood is usually arterial, bright red, and frothy; in hæmatemesis it is black and clotted. But if large vessels in the stomach have ruptured, the blood may also present arterial characteristics, while in hæmoptysis, on the other hand, it may appear blackish-red and clotted if it has remained in a cavity for a certain length of time. *c.* In hæmoptysis the blood is alkaline, in hæmatemesis it has an acid reaction. But if the gastric hemorrhage is very profuse, the gastric juice may be unable to change the normal reaction of the blood so that it remains alkaline or neutral. *d.* The admixture of large amounts of food with the blood is indicative of hæmatemesis, but the difficulties mentioned under *a* must be taken into consideration. *e.* The passage of a black (bloody) stool some time after the hemorrhage is indicative of gastric hemorrhage. *f.* The clinical history points to disease of the respiratory organs in hæmoptysis. *g.* The objective changes in the organs must be taken into consideration.

It must be determined, in addition, whether the hæmoptysis is bronchial or pulmonary in its origin. This depends mainly on the etiology.

Finally, it is very difficult to decide whether the hemorrhage is arterial or capillary, but this question may be left open since it possesses no special practical significance.

V. PROGNOSIS.—As a rule, the prognosis of hæmoptysis *per se* is not unfavorable. The profuse hemorrhage may cause death from suffocation (by occlusion of the bronchi), or from the mere loss of blood, but this occurs in extremely few cases. The prognosis is unfavorable if the hemorrhage is the result of pulmonary apoplexy. The hæmoptysis often assumes a grave significance on account of the serious nature of the primary disease.

VI. TREATMENT.—Hæmoptysis may be prevented at times by prophylactic measures. Patients who suffer from acute or chronic inflammatory processes of the lungs should be guarded against cold or violent exertion; severe cough should be treated with narcotics. In cases of heart disease, digitalis should not be used to excess. It is said that this drug favors the separation of cardiac thrombi and the formation of hemorrhagic infarctions. If marantic thrombi have formed in the peripheral veins, the limb should be kept as quiet as possible and not handled in the vicinity of the thrombus. Furuncles of the skin are treated with ice compresses in order to prevent the formation of thrombi. Women should not be allowed to get up too early from child-bed, since a con-



considerable number of the sudden deaths in this condition are the result of embolism of one of the larger branches of the pulmonary artery, secondary to thrombosis of the veins of the leg or the genital apparatus.

If the hæmoptysis has begun, the patients should at once be put to bed, and all movements of the body and speaking should be strictly interdicted. At first the physician should not make a thorough examination, as this is apt to increase the hemorrhage. It must be remembered, also, that the therapeutic indication is the same in all forms of hæmoptysis.

The patient should be permitted to take nothing but cold fluids, for example, iced milk or ice water. An ice-bag is applied over the supposed site of the hemorrhage, and small pieces of ice may be swallowed. Ergotin may be injected every morning and evening under the integument of the thorax. If there is an intense desire to cough, narcotics must be administered (morphin, hydrochloric, 0.005 every three hours, or opium, 0.02 every two hours). The patient should be directed to cough vigorously and an expectorant may be administered only when the bronchi are occluded and there is danger of suffocation.

If there is no other remedy at hand, the patient may swallow one or two teaspoonfuls of salt. In some cases this remedy always checks the hemorrhage so that the aid of the physician is unnecessary. Heller obtained good results with bromide of potassium (1.5 t. i. d. in water). Numerous astringents have also been employed, such as acetate of lead (0.05 with opium, 0.02 every two hours), tannic acid (0.2 every two hours), alum, rhatany, liq. ferri sesquichlorati, gallic acid, etc.

Astringents are often used in the form of inhalations (solutions of sesquichloride of iron, alum, or tamin). We are not very enthusiastic advocates of this method of treatment. Cases have been reported in which chronic inflammations made rapid progress as the result of such inhalations.

The balsams are recommended by many writers, and balsam of copaiba has recently found a warm advocate in Almér.

Massina and Peter report prompt effects from the administration of emetics. Ipecac is specially recommended among remedies of this class.

Mineral acids are often used in this affection. If the action of the heart is very much accelerated, digitalis may be administered.

The action of internal remedies is occasionally aided by that of external irritants (chloroform inunctions to the chest, cups, vesicants, leeches). Oppolzer recommended a small venesection in constantly recurring hemorrhages.

In the hæmoptysis of intermittent fever, large doses of quinine should be given (1.0–2.0 four hours before the attack is expected).

Stimulants are indicated when the vital energies begin to fail.

After the cessation of the hemorrhage, care should be taken to prevent decomposition of the mass of blood remaining within the lung. The air of the room should be pure and disinfected with the carbolic spray.

## 2. *Alveolar Emphysema of the Lungs.*

I. ANATOMICAL CHANGES.—Alveolar emphysema of the lungs depends on dilatation of the infundibular alveolar spaces, associated with disappearance of the interalveolar and interfundibular septa. Simple ectasia of the infundibula and alveoli (dilatation of the lungs) constitutes to a certain extent a prodromal stage of fully developed emphysema.

But not every ectasia leads to emphysema; if the causes of the former condition are temporary in character, the status quo ante may be restored. If acute dilatation of the lungs is constantly repeated, it will finally lead to emphysema.

The disease may be unilateral or bilateral, circumscribed or diffuse. The affected parts are pale, rosy, anæmic and dry, and creak very slightly or not at all under the pressure of the finger. On palpating the parts, we obtain the sensation of a tense air cushion. Upon cutting through the emphysematous parts, they collapse and allow us to recognize large air spaces, varying from the size of a pin's head to that of a pigeon's egg. As a matter of course, the largest air spaces result from the fusion of dilated infundibula.

Emphysema develops with special frequency at the anterior median and lower borders of the lungs which appear swollen and rounded. In like manner the lingual process of the left lung very often presents the emphysematous change. It is observed most frequently in the upper lobes, particularly upon the anterior convex surface. It almost always affects the peripheral layers of the lung, and rarely extends deep into the organ. This is also true of dilatation of the lung.

The following appearances are presented in long standing and extensive emphysema: the thorax presents a striking curvature and is barrel-shaped; the costal cartilages are thickened and partly calcified. The median edges of the lungs cover the pericardium completely, or almost completely, the left border passing towards the median line until it is almost in contact with the right border. The lower border of the lung and the diaphragm are abnormally depressed. The lungs collapse very little on opening the thorax; if there is catarrh of the finer bronchi, the lungs may even project from the thorax.

In addition to emphysema, the lungs usually present other changes, such as bronchitis, pneumonic infiltrations, processes of retraction, adhesions.

The heart is situated lower than normal, has a more horizontal position, and the right ventricle and auricle are principally directed to the front. These parts of the organ are distended with blood. The right ventricle and auricle are dilated and hypertrophied; the hypertrophy is especially noticeable at the conus pulmonalis. The muscular tissue is sometimes brownish-red, sometimes streaked with yellow, the latter being the result of fatty degeneration.

The venæ cava contain a large amount of blood which is usually of a black color. The spleen presents evidences of stasis (slight enlargement, proliferation of the trabecular connective tissue). The liver often presents similar appearances—at first venous congestion and enlargement, later diminution in size and retraction (cyanotic, atrophic nutmeg liver).

Swelling and catarrhal changes of the gastro-intestinal mucous membrane are found. The hemorrhoidal veins are not infrequently dilated. The kidneys present evidences of venous stasis, followed, at a later period, by processes of retraction.

Microscopical examination shows that the entire process begins with dilatation of the infundibular spaces, and that this is followed by ectasia of the alveoli. The intercapillary portions of the alveoli suffer the first dilatation. The more the latter enlarge, the more the septa between the alveoli must diminish in height. At the same time the septa undergo atrophy on account of the tension to which they are subjected.

They become very thin, are perforated and partially destroyed, so that several alveoli coalesce into one space, and finally the many-chambered infundibulum is converted into a single chamber; at first this is pyramidal, but later it becomes spheroidal.

Finally, the network of elastic fibres which are situated in the septa between the infundibula may also atrophy and disappear, so that two or more infundibula coalesce into one.

The alveolar epithelium cells generally contain an accumulation of fat granules near the nucleus—changes which precede the final disappearance of the cells. A similar condition is also noticed in the endothelium of the blood-vessels and the connective-tissue cells of the stroma of the lung which finally disappear in the course of pulmonary emphysema.

According to Eppinger, the finest elastic fibres are the first to disappear so that the elastic network assumes a much simpler structure. The coarser fibres are not affected until a later period. Eppinger believes that these changes are the result of mechanical stretching.

By means of silver staining Isaaksohn found that the endothelial markings of the pulmonary capillaries were absent in places, the vessel presenting a finely granular appearance. An accumulation of white blood-globules and the formation of a thrombus were noticed at such localities. The occluded vascular districts undergo fatty degeneration and finally disappear.

The vessels are then found to be scanty and have an abnormally straight course. Isaaksohn regards these vascular changes as the starting-point of emphysema. At all events they must exert a decided influence on the respiratory process and also on the circulation in the pulmonary artery. Rindfleisch showed that new channels are opened in the place of the vessels which have been destroyed and that, among others, the pulmonary arteries enter into more intimate connection with the bronchial veins. Rindfleisch describes hypertrophy of the smooth muscular fibres in the walls of the fine bronchi, but Colberg maintains that these structures undergo atrophy.

In senile atrophy the lungs present changes which are similar, to a certain extent, to those found in alveolar emphysema, but are distinguished from the latter by the fact that the stage of dilatation of the lungs is wanting. The process begins with atrophic changes in the alveolar and later in the infundibular septa. But on account of the absence of the stage of dilatation, the affected lung is smaller than normal.

II. ETIOLOGY.—Vesicular emphysema is mainly a disease of advanced age; it is rare in childhood and youth. It is also more frequent in men than in women. Climatic influences also exert an undoubted effect; it is more frequent in Northern, cold climates than in warm regions of a more uniform temperature.

Certain writers attach etiological significance to heredity, and there is no doubt that families are occasionally met with in which grandparents, parents, and children suffer from pulmonary emphysema. But it seems to us that the patients do not inherit a predisposition to emphysema, but rather to diseases associated with cough, and that these give rise secondarily to emphysema.

The term congenital emphysema is sometimes improperly used. This term refers to certain hypoplastic (not retrogressive) and atrophic



conditions which have led to the development of large infundibular and alveolar spaces.

Emphysema rarely develops without previous disturbances in the mechanism of respiration, and affections of the respiratory apparatus. The most frequent cause is catarrh of the finer bronchi. Stenoses of the bronchi (the result of thyroid enlargement, foreign bodies, fibrinous exudations and the like), are also regarded as causes of emphysema.

Long-continued and repeated coughing and straining also constitute a widespread cause of the disease. This is observed in whooping-cough, playing on brass instruments, lifting heavy loads, chronic constipation, continued screaming and singing. Distention and emphysema of the lungs have also been noticed after straining in difficult labor, spasms of laughter, attacks of asthma, etc. Similar conditions are produced in asphyctic new-born infants, as the result of the too vigorous blowing-in of air while performing artificial respiration. The conditions mentioned may also follow the inhalation of irritating gases.

The form of emphysema referred to above is known as substantive emphysema to distinguish it from vicarious or compensatory emphysema. The latter form develops when some parts of the lungs are excluded from respiration to a greater or less extent, and the other parts endeavor to assume their function by an increase in size. This is observed in many affections of the pulmonary parenchyma, in compression of the lungs by pleuritic exudation, pericarditis, excessive dilatation of the heart, aneurisms of the heart and pulmonary artery, spinal curvature, etc. This category also includes that form of emphysema which is found not infrequently next to pleuritic adhesions.

Both forms of emphysema may be present at the same time. If there is extensive catarrh of the finer bronchi, the region of the catarrhal affection furnishes the conditions for substantive emphysema, while vicarious emphysema may develop in the unaffected portions of the lungs.

The mechanical theory of emphysema maintains that sometimes the forced inspirations, sometimes the disturbances of expiration give rise to the emphysematous process. According to the nutritive theory, the disease is the result of nutritive disturbances of the pulmonary tissue (hyperplasia of the connective-tissue cells, according to Villemin; disease of the vessels, according to Isaaksohn). According to Freund, emphysema is the result of hypertrophy with subsequent ossification of the costal cartilages; by this means the thorax undergoes primary rigid dilatation, to which the lungs must accommodate themselves.

As a matter of fact, however, the disease sometimes develops in one way, sometimes in another.

With regard to the nutritive theory it must be remarked that it has not proven hitherto that the nutritive disturbances are sufficient to give rise to vesicular emphysema. As a matter of course, they will favor the development of emphysema as soon as mechanical disturbances of respiration make their appearance, and the latter require much less intensity in order to produce emphysema than if they are not associated with the former. Clinical experience teaches that certain injurious influences to which patients have been previously subjected without bad effect, will suddenly give rise to emphysema after inflammatory or other changes in the pulmonary parenchyma have developed.

Vicarious emphysema, the emphysema in catarrh of the finer bronchi and in stenosis of the bronchi by foreign bodies, are mainly the effect

of disturbances of inspiration. If a large part of the lung is excluded from respiration, the normal amount of inspired air must be collected into a smaller space, in other words, the latter must undergo dilatation. These conditions obtain most clearly if certain bronchi and alveoli are occluded. They also obtain in pleuritic adhesions in which the adjacent lung does not undergo sufficient expansion during inspiration on account of its fixation, and the freely movable portions of the lung (usually the anterior and lower borders) undergo compensatory emphysematous changes.

The inspiratory disturbances are different in catarrh of the finer bronchi or in stenosis of the bronchi. Here the inspiratory current of air gains access to the alveoli, but during expiration very little or no air leaves the alveolar space on account of the valve-like occlusion of the finer bronchi. Hence distention and later emphysematous changes are the necessary consequences.

The forms of emphysema which result from disturbances of expiration include all those in which spells of coughing and straining take a part. In coughing or bearing down when the glottis is closed, the chief pressure is experienced by the lower portions of the lungs. Hence the expiratory current of air flows from these parts into the trachea, but as it meets with an obstruction at the closed glottis it is forced back, in part, into the main bronchus of the upper lobe, causing this part of the lung to dilate and, at a later period, to undergo the emphysematous change.

That the upper part of the lung is most affected under such conditions was demonstrated by Ziemssen upon an otherwise healthy man, whose pectoral muscle was wanting, and in whom the effects of coughing and straining could be followed through the thin intercostal muscles.

But the most frequent form of emphysema—that which follows long-continued catarrh of the finer bronchi—shows that all the modes of development of the disease may be combined with one another. In the region of the inflamed bronchi, inspiratory emphysema develops; in the free districts, vicarious inspiratory emphysema may develop in another manner. The usually very obstinate cough gives rise to expiratory emphysema. Finally, the catarrhal condition will affect the nutrition of the pulmonary tissue, make it less resistant, and thus favor the development of emphysema.

III. SYMPTOMS.—The disturbances which give rise to alveolar emphysema are, in the first place, of a mechanical character, and are manifested directly by changes in the mechanism of the respiration, and in the circulation of the blood.

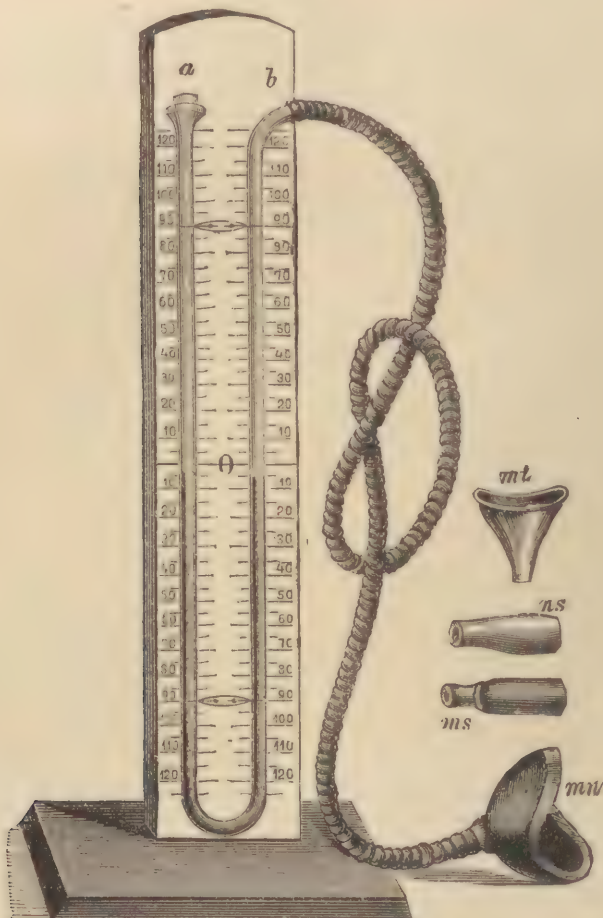
When a considerable part of the elastic tissue contained in the pulmonary stroma is destroyed, as the result of the emphysematous process, the elasticity of the lung, in other words, its tendency to contract, is diminished. As this comes into play mainly during expiration, emphysema will chiefly affect this part of the respiratory act. This can be demonstrated with the aid of Waldenburg's pneumatometer, or Riegel's stethography.

The pneumatometer is a U-tube filled with mercury, and graduated in millimetres. One leg is connected by a rubber tube with a mouth-piece or nose-piece. After a deep, free inspiration the patient expires through the mouth-piece, or, after a deep expiration, inspires through the "mouth-nose" piece (suction must be avoided). The in-

spiratory and expiratory pressure is read off by doubling the number of millimetres which the mercury, in the tube nearest the patient, has fallen below 0 during expiration, and has risen above 0 during expiration. By means of this instrument, it is found that the expiratory pressure is unusually small, being even less than that of inspiration (normal expiratory pressure is 30–100 mm. Hg.).

Stethographic examination shows that the expiratory portion of the

FIG. 62.



Pneumatometer. After Waldenburg. *mn*, mouth and nose mask; *ms*, mouth piece; *ns*, nose piece; *mt*, mouth funnel.

respiratory curve is abnormal, and falls off abruptly (Fig. 63). Marly and Pick have shown that the respiratory curve in emphysema is very similar to that obtained in animals whose cervical pneumogastrics have been cut.

As a matter of course, the imperfect expiration interferes with the pulmonary interchange of gases, and thus causes dyspnoea.

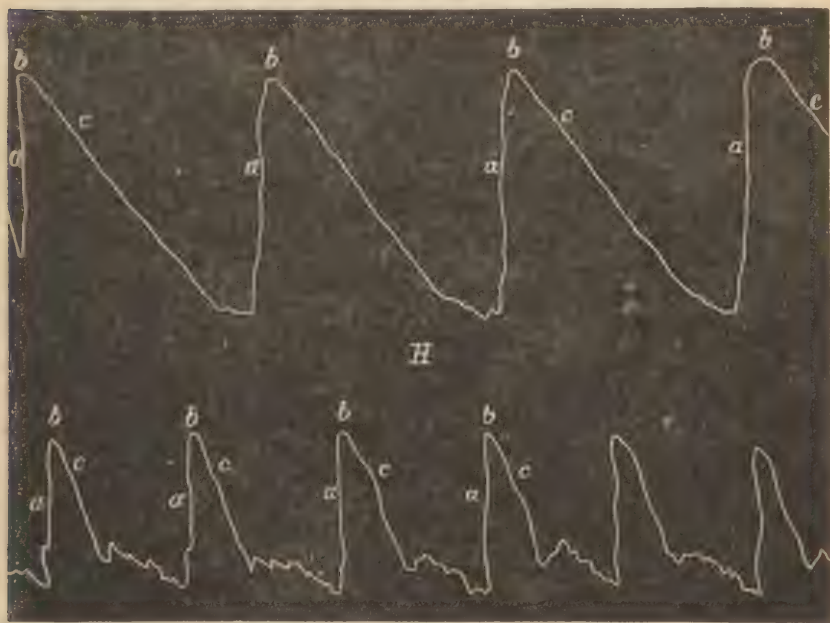


It also acts unfavorably upon the circulation in the domain of the pulmonary artery. In addition, the destruction of a large number of pulmonary capillaries diminishes the surface upon which the interchange of gases takes place between the blood and atmospheric air.

The entire pulmonary circulation would be irreparably disturbed did not the heart possess the power of compensating, to a certain extent, the circulatory disturbances. As the destruction of pulmonary capillaries increases the pressure in the pulmonary artery, dilatation and hypertrophy of the right ventricle are necessary physical consequences. But when the energy of the cardiac muscle becomes impaired, symptoms of stasis appear, and proceed to a fatal termination.

The disturbances of respiration and circulation aid one another in giving rise to the feeling of want of breath. If emphysema is uncom-

FIG. 63.



Stethographic curve of the diaphragm. After Riegel. I., normal curve; II., curve of emphysema. *a*, ascending inspiratory curve; *b*, transition between the inspiratory and expiratory curves; *c*, descending expiratory curve.

plicated and not very severe, the dyspnoea will appear only when the patient performs active movements. The dyspnoea becomes excessive if bronchitis develops, or an existing catarrh grows more severe (usually in the spring and autumn). Asthmatic symptoms are observed not infrequently under such circumstances.

The signs on percussion are the most important ones in physical diagnosis. They consist of permanent lowering of the lower borders of the lungs, slight or absent movement of the borders of the lungs during respiration, and permanent diminution or disappearance of cardiac dullness.

A probable diagnosis may be made in some cases by mere inspection. The thorax is very much dilated and rounded. The antero-posterior

diameter is mainly increased in size, but the transverse and oblique diameters are also enlarged. The sternum has a more marked curve anteriorly, the spine is more strongly curved posteriorly. The dilatation is sometimes confined to the upper, but particularly to the middle portion of the chest (barrel-shaped thorax), sometimes it is general (Fig. 64). The cyrtometer curve approaches the shape of a circle rather than that of an ellipse (Fig. 65).

The intercostal spaces are usually widened, and the lower ones are alone recognizable as shallow grooves. The supraclavicular fossæ are flattened, and occasionally they even project externally. In some cases,

FIG. 64.



Barrel-shaped thorax of pulmonary emphysema. After Rehn.

severe cough gives rise to a protrusion of the supraclavicular fossæ, corresponding to the apex of the lung. In one case, Friedreich observed a hernial protrusion of the lung, as large as a hen's egg, in the fifth right intercostal space.

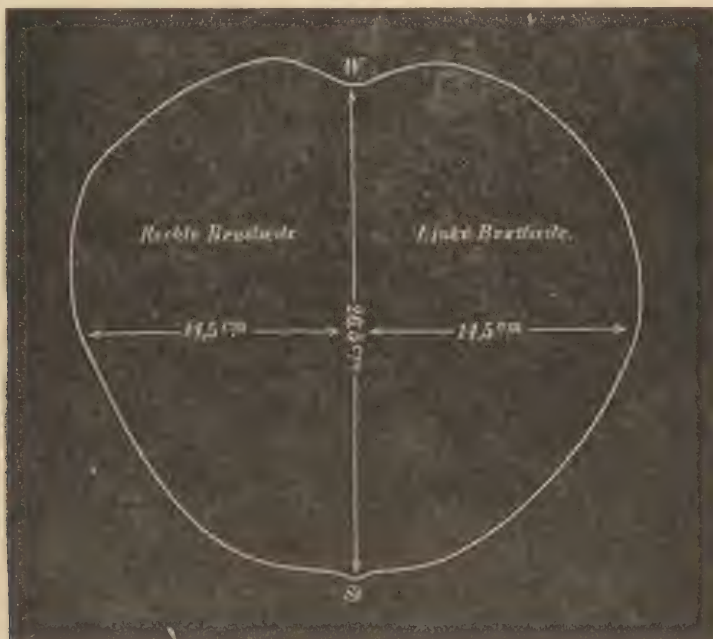
This should not be mistaken for the expiratory swelling of the bulbus of the internal jugular, which is observed not infrequently in emphysema if circulatory disturbances are present, and occasionally attains the dimensions of a hen's egg.

The frequency of the respirations is almost always increased. The respiratory excursions of the thorax are very slight, and even at the end of expiration the chest seems, on superficial examination, to be still in the inspiratory position (permanent inspiratory thorax). In contrast to

the slight respiratory excursions of the thorax is the vigorous action of the auxiliary muscles of respiration. The sterno-mastoids and scaleni soon undergo hypertrophy, and project as tense cords beneath the integument of the neck.

The skin and mucous membranes usually present the signs of more or less marked cyanosis. This may acquire great intensity if the emphysema is complicated with bronchitis and failure of the heart (symptoms of stasis).

FIG. 65.



Cyrtometer curve of an emphysematous thorax in a man *æt.* 46 years.  $\frac{1}{2}$  natural size. *W*, vertebral column; *St*, sternum.

Venous undulation and venous pulse occur under such circumstances in the veins of the neck. Epigastric pulsation is observed not infrequently in emphysematous patients.

Emphysema is not always associated with dilatation of the thorax; indeed, it is sometimes found in the phthisical thorax. The development of the emphysematous chest is dependent on the primary cause of the emphysema, and also upon the yielding character of the thorax. It follows the loss of elastic fibres in the pulmonary tissue, for the traction permanently exerted by the lungs upon the inner surface of the thorax diminishes with the decreasing elasticity of the pulmonary tissue. The emphysema is sometimes unilateral, and in such cases the dilatation of the thorax will occur upon the affected side.

Palpation generally reveals increased thoracic resistance. The costal cartilages, particularly the upper ones, are thickened, elongated, rigid, and ossified. Upon the application of the hands to the chest we can convince ourselves of its slight excursive capacity. Vocal fremitus may be distinctly diminished on account of the slight power of vibration of the chest-walls. The spirometer shows diminished vital capacity of the lungs.



The apex beat is usually not perceptible. The movements of the heart cannot be felt at all, or they are felt very feebly. At each systole we can often feel, immediately above the ensiform cartilage, a deeply-situated body which grows hard and projects downward. This corresponds to the lower border of the right ventricle.

The percussion sound is often characterized by its striking loudness and low pitch. Over the lower posterior and lateral parts, more rarely over other regions, it often sounds as if an empty pasteboard box is percussed. Pulmonary resonance extends lower than normal. On the anterior surface of the right thorax the upper lesser (absolute) liver dulness begins in the right mammary line below the eighth rib, occasionally at the lower border of the thorax. The movements of the lower border of the lung are either abolished or very slight during deep inspiration. On the left side the cardiac dulness has diminished or disappeared. Traube's semilunar space may also be diminished in height, and likewise presents slight or absent mobility of the edges of the lungs. On the posterior surface of the thorax the lower border of the lung is found at the level of the spinous process of the twelfth dorsal vertebra, and here likewise the respiratory mobility is absent or very slight.

Da Costa and Friedreich found that the respiratory change in pitch, *i. e.*, the differences in the percussion note during inspiration and expiration, was indistinct in slight grades of emphysema, and disappeared in severe forms. Delafield describes a wooden percussion note, and Thompson states that on gentle percussion he has heard a muffled high-pitched note which, in doubtful cases, permits the differential diagnosis from miliary tuberculosis.

Auscultation usually reveals enfeebled respiratory murmur, and occasionally the latter is entirely inaudible.

The heart sounds are very feeble and occasionally disappear (projection of the lung over the heart). The second (diastolic) pulmonary sound is usually intensified, indicating increase of pressure in the pulmonary artery. Cardiac murmurs are heard not infrequently; they are almost always of a systolic character, and are heard generally at the apex or over the tricuspid valves.

These murmurs are sometimes anæmic, sometimes the result of changes in the heart muscle, particularly fatty degeneration. If the cardiac dilatation becomes excessive, the tricuspid, more rarely the mitral valves become relatively insufficient. Ganghofner has also heard diastolic murmurs over the lower part of the sternum and at the apex.

The physical signs are often complicated by intercurrent conditions.

Emphysema may last a very long time. Patients who were attacked by the disease during childhood sometimes attain the age of 60 years or more. Death takes place most frequently from heart failure, either because the work to be performed by the heart is suddenly increased by a complicating bronchitis, or by reason of fatty degeneration of the cardiac muscle. Symptoms of stasis then develop, starting from the right heart and extending into the *venæ cavæ*.

We will give a mere sketch of these symptoms, which are identical with those occurring in primary disease of the heart. The scene generally opens with œdema of the ankles, which at first disappears during the night, and on prolonged horizontal decubitus. The œdema soon becomes constant and extends to the legs. Ascites develops; diuresis becomes scanty; the urine is very acid, of high specific gravity, contains a good deal of uric acid, also albumin and casts. Then vomiting, anorexia,

diarrhœa, even intestinal hemorrhages make their appearance. Hydrothorax and hydro-pericardium develop; the dyspnœa becomes excessive; the sensation of a rush of blood to the head, dizziness, difficulty of hearing, and spots before the eyes testify to cerebral hyperæmia. The face is often swollen into an unshapely mass, is extremely blue, and the eyes may protrude from their sockets.

In one case Litten observed prominence of the optic papilla, numerous irregular or nummular hemorrhages along the retinal veins and neuroretinitis.

If the symptoms of stasis become extreme, death from suffocation may take place. In some cases death is the result of œdema of the lungs, in others symptoms of cerebral hemorrhage make their appearance.

Intercurrent affections may be a source of great danger. Stokes had noticed that an emphysematous spot, situated at the periphery, occasionally ruptures and gives rise to pneumothorax. This accident is generally preceded by a violent coughing spell or great bodily exertion, but it may occur spontaneously during sleep. Emphysema of the skin may be produced also from previous rupture of the pulmonary tissue, and the development of interstitial emphysema of the lungs. Profuse pulmonary hemorrhages sometimes, though not often, are observed if very violent catarrh is present. Pulmonary hemorrhage is sometimes the result of hemorrhagic infarction, the latter being produced by the separation of thrombi from the dilated right ventricle.

It was formerly taught that emphysema antagonizes the development of phthisis and of valvular lesions of the heart. This doctrine is only true in general, exceptions to it being found not infrequently.

IV. DIAGNOSIS.—Slight grades of vesicular emphysema remain unrecognized during life. A probable diagnosis may be made at an early stage if dyspnœa, the cause of which cannot be explained, is present, and at the same time the pneumatometer shows extremely slight expiratory pressure.

The following conditions must be considered in making a differential diagnosis:

*a.* Acute dilatation of the lungs.

The differential diagnosis cannot always be made at the first examination, unless there is a history of long-standing cough. The recovery of the respiratory affection, while the borders of the lungs remain depressed, favors the diagnosis of emphysema.

*b.* Congenital hypertrophy of the lungs.

In this affection, the lower borders of the lungs are lower than normal, on account of their greater volume, but they retain their **respiratory mobility**.

*c.* Pneumothorax.

This disease is almost always unilateral, and the sounds heard on **percussion** and auscultation present a metallic quality, which is absent in emphysema. Furthermore, pneumothorax develops acutely.

*d.* Aneurism.

Biermer describes a case in which he mistook an aortic aneurism for pulmonary emphysema. Our attention should be directed to abnormal vascular murmurs or sounds, and to the character of the pulse.

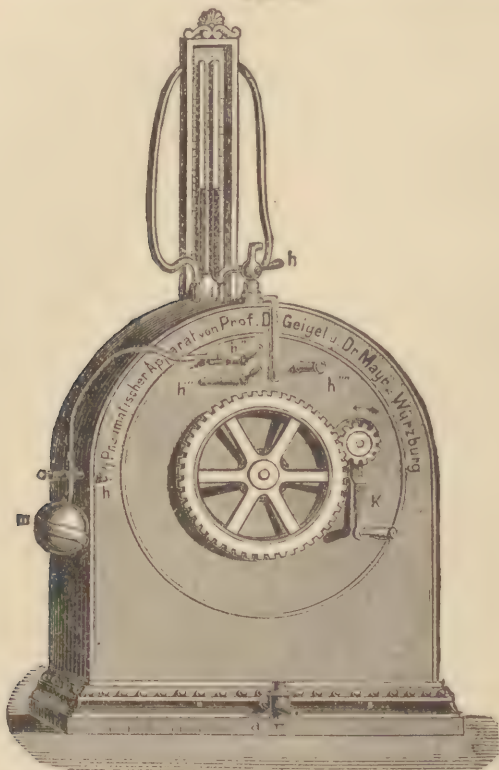
V. PROGNOSIS.—The prognosis is not very unfavorable, so far as danger to life is concerned. Life may be prolonged many years, despite the existence of a moderate degree of emphysema. The prognosis, as regards recovery, however, is unfavorable.

The prognosis depends, to a certain extent, upon the ability of the patient to rid himself of any injurious influences which may be acting upon him. Obstinate and extensive bronchitis also aggravates the prognosis, because a greater amount of work thereby devolves upon the heart. The power of the heart muscle is an essential element in the prognosis, since its impairment leads inevitably to symptoms of stasis.

Certain forms of emphysema must be regarded as favorable events, for example, vicarious emphysema, which alone may render the respiratory process possible.

VI. TREATMENT.—Prophylaxis should not be left out of consideration in the treatment of emphysema. Bronchial catarrh should be care-

FIG. 66.



Ratchet-wheel ventilator (Geigel and Mayr). 1/10 natural size.

fully treated. The patients should avoid excessive, unnecessary exertion in making the respiratory movements.

Pneumato-therapeutics constitutes the chief method of treatment of emphysema. As expiration is mainly disturbed, expiration into rarefied air seems the most rational procedure, and this view is confirmed by the therapeutic results. If the patient is also suffering from catarrh of the finer bronchi, the inhalation of compressed air is likewise indicated. Either the compressed air is first inhaled, and later the patient expires into rarefied air, or, by a combination of two apparatuses, both methods are carried out alternately. Two or three sittings should

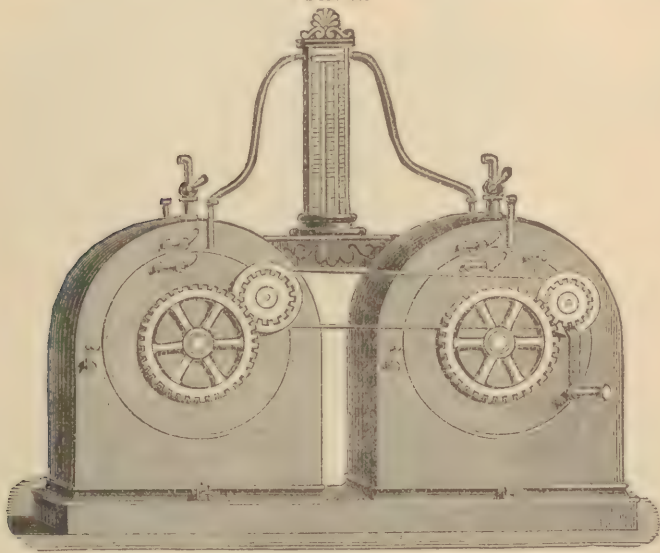


be held daily, their duration being about ten minutes in the beginning, and later increased to a half or even a whole hour.

Pneumatic apparatus is classified as portable apparatus, and pneumatic cabinets.

Among the portable apparatus, the most perfect, but at the same time the most expensive and least portable, is Geigel and Mayr's ratchet-wheel ventilator. This consists of a sheet-iron cage-vessel, within which a so-called ratchet-wheel, which is partly under water, may be revolved by means of a crank placed on the outside (vide Fig. 66, *k*). Within the apparatus, and above the ratchet-wheel, is a bell-shaped space to receive the air which has been carried under water by the wheel. Both spaces of the apparatus are connected by the stop-cocks  $h'-h'''$  with the external air, the cage-like space communicating with  $h'$  and  $h''$ , the bell space with  $h'''$  and  $h''''$ . The cock  $h'$ , which can be unscrewed,

FIG. 67.



Double ventilator (Geigel and Mayr).

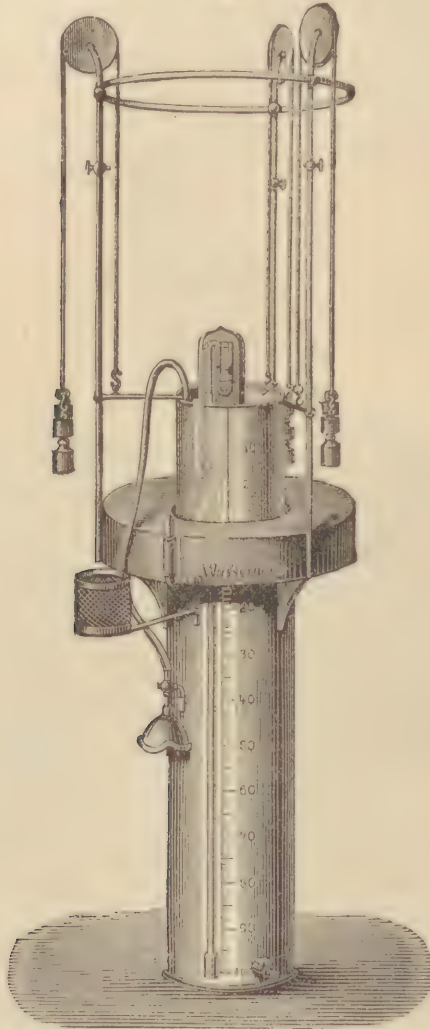
serves to fill the apparatus with water,  $a$  gives exit to the water; when the apparatus contains sufficient water, it begins to flow from  $p$ .

If  $h'$  and  $h''$  are open,  $h'''$  and  $h''''$  closed, the revolution of the crank, which turns in the direction of the arrow, will force air from the outer cage into the bell-shaped space, and, as the air cannot escape, it is compressed more and more at each revolution. If  $h'''$  is connected with a mouth-nose mask ( $m$ ) by a rubber tube, which by means of a valve is brought into communication, according as the valve is turned, with the external air, or with the rubber tube, and, by means of  $h'''$ , with the inside of the bell space, it evidently becomes possible to inspire the compressed air. By continued revolution of the crank, the pressure of the compressed air may be kept at the same level. If  $h'$  and  $h''$  are closed, and  $h'''$  and  $h''''$  are opened, the air in the outer cavity will be pumped by the ratchet-wheel into the bell cavity.

From the latter it may escape through  $h'''$  and  $h''''$ . In the outer

space, on the other hand, rarefaction of the air occurs, and if  $h'$  is connected, by a tube, with the mouth-mask, it becomes possible to expire air into a space filled with rarefied air. The degree of compression or rarefaction is shown by a water-manometer, one leg of which is connected with the bell space, the other with the outer space. The

FIG. 68.



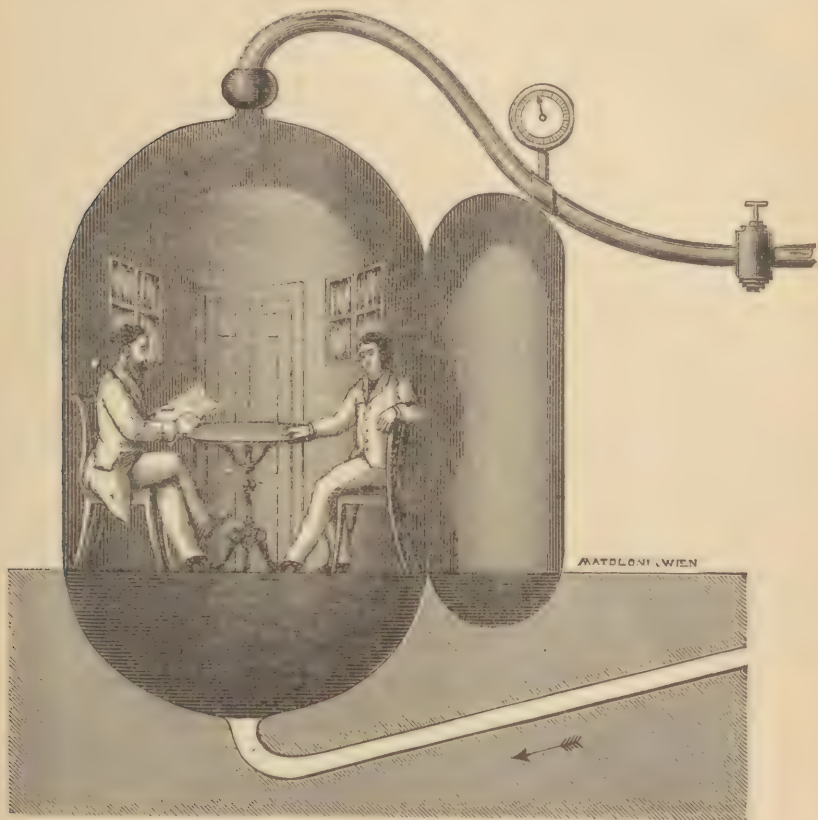
Waldenburg's portable inhalation apparatus. 1/15 natural size.

column of water in the manometer is marked in centimetres, next to which is the equivalent in atmospheric pressure. Experience teaches that changes of one-fortieth to one-thirtieth of the atmospheric pressure are the most effective.

As a matter of course, a single apparatus only permits inspiration of

compressed air, or expiration into rarefied air, or intermittent breathing. If the patient is to carry out alternate breathing, *i. e.*, expire into rarefied air and inspire compressed air alternately, a double ventilator becomes necessary (Fig. 67). This is merely a combination of two ratchet-wheel ventilators, whose wheels are turned by a single crank. The cocks *h'* and *h''* of the mantel-shaped space in the one apparatus are opened in order to produce compressed air, while in the other, the cocks *h'''* and *h''''*

FIG. 69.



Pneumatic cabinet. After Tabarié.

of the bell space are opened in order to produce rarefied air in the mantel-shaped space.

Waldenburg's apparatus is of older date but less perfect (Fig. 68).

This consists of two metal cylinders, the smaller of which moves up and down within the larger one. The outer cylinder is filled with water, the amount being read off upon a manometer. The inner one is connected by a rubber tube with a mouth-nose mask, which, by means of a valve, may be connected with the external air, or the inner cylindrical space. If the inner cylinder is standing on the bottom of the outer one, and weights are attached to the hooks which are connected with the top



of the inner cylinder, the latter will be drawn upwards and the air within it rarefied. If the patient breathes rhythmically into the inner space, he expires into rarefied air. If the inner cylinder is drawn up as far as possible and weights placed upon its upper surface, the air in the cylinder will be compressed and can be inspired. The degree of compression or rarefaction is measured by a mercury manometer.

The mechanical action of the air in pneumatic cabinets differs from that of portable apparatus, since it is not confined to the lungs, but extends over the entire surface of the body. As a rule, only compressed air is employed, so that the patient inspires compressed air and expires into it. In the beginning, the apparatus should be used one hour, then one and a half to two hours.

The oldest apparatus, viz., that of Tabarié, was shaped like a diver's bell. It is an ellipsoidal body, made of sheet iron, which will hold four to twelve people. The lower third of the cabinet is situated below the ground. Above the base is a horizontal perforated floor; the doors open from the inside. Below is the pipe for the entrance of air, above is one for its exit. The air is pumped in by means of a steam engine. If, by means of a stop-cock in the upper pipe, less air is allowed to escape than enters below, the air in the cabinet will be compressed, the degree of compression being shown by a manometer.

Pneumatic cabinets have been recently perfected by V. Liebig, Simonoff, and others.

Gerhardt recommends rhythmical compression of the thorax during expiration, in order to facilitate this portion of the respiratory act. This is done two or three times a day during twenty to thirty respirations. In two cases this was followed by an increase in the vital capacity of the lungs, but slight bronchial hemorrhages occurred, and, in one case, twitchings in the face.

Dobell recommended the retardation of inspiration as a plan of treatment. He constructed a mouth-piece which impeded inspiration but allowed free expiration, and directed the patient to breathe with the nostrils closed.

Complications and the primary diseases must be treated according to general principles.

### 3. *Interlobular (Interstitial) Emphysema of the Lungs.*

1. In interlobular emphysema the air passes into the interfundibular and interlobular connective tissue, and particularly into the lymph channels. This condition is inconceivable without previous rupture of the alveolar walls. It is found most frequently in the anterior parts of the upper lobes.

In corpses which are undergoing decomposition, gas may develop spontaneously in the interstitial connective tissue of the lungs.

The air which enters the interstitial tissue during life rarely remains at the site of lesion, but passes to the periphery of the lung, into the subpleural connective tissue. The pleura is raised into small transparent vesicles which are arranged like a rosary, and are grouped into fields which correspond to the boundaries of one or more lobules. The vesicles may be pushed along these boundaries by the pressure of the finger, thus serving to distinguish them from the subpleural vesicles of alveolar emphysema. The pleura is rarely raised into large vesicles. Bouilland mentions a case, however, in which the pulmonary pleura at the base of

the left lung was elevated into a sac, which was so large as to be mistaken at first for the stomach.

The air may also pass along the connective tissue to the hilus of the lung, and thence to the mediastinum and the cellular tissue of the neck, thus giving rise to subcutaneous emphysema of the neck, or even the larger part of the body.

2. Interstitial emphysema may follow the alveolar form, if violent cough, continued straining, or other similar disturbances cause rupture of the atrophied walls of the alveoli. Interstitial emphysema is observed most frequently under the following circumstances:

*a.* After continued screaming.

*b.* After severe coughing and straining, as in pertussis, capillary bronchitis, or lifting heavy weights. Haultœur collated twelve cases in which the condition developed during difficult labor.

*c.* From stenosis of the air passages by foreign bodies, mucous, blood, or fibrinous exudation. This condition has also been observed a number of times in croup.

*d.* From ulcerative processes in the pulmonary parenchyma, for example, pulmonary phthisis.

*e.* As the result of injuries. Holthouse and Adams report a case in a child who was run over without sustaining any other injuries. In this instance the emphysema was probably the result of excessive compression of the lungs, the glottis being closed at the time. In the asphyxiated new-born, this condition may result from the violent blowing of air into the larynx in maintaining artificial respiration.

3. Interstitial emphysema of the lungs can rarely be diagnosed during life. We are only able to make the diagnosis if emphysema of the skin develops, and this, as was shown by Traube, must first appear in the jugular fossa. Other causes must be excluded (injuries of the trachea, larynx, and œsophagus). The cutaneous emphysema is recognized by the swelling, absence of folds, and gloss of the skin; it is very pale, of an alabaster color. The integument has a crackling feel, and after pressure with the finger, a depression is left, which soon disappears. Compression of the large vessels of the heart as the result of mediastinal emphysema may give rise to serious conditions of dyspnoea; at the same time, the jugular veins become swollen.

4. As a rule, cutaneous emphysema disappears spontaneously. If dyspnoea is noticeable, it may be relieved by the administration of narcotics, which, at the same time, moderate the cough, and thus prevent renewed entrance of air.

#### 4. *Pulmonary Atelectasis.*

(*Collapse of the Lungs. Apneumotosis.*)

I. ETIOLOGY.—Atelectasis signifies disappearance or diminution of the amount of air in the pulmonary alveoli. It may be divided etiologicaly into four forms, viz., congenital, obstruction or absorption, compression, and marasmic atelectasis.

Congenital atelectasis is not a newly developed condition. Before birth the lungs are atelectatic, and are not distended with air until the beginning of respiration. If the mechanism of respiration is disturbed, more or less extensive portions of the lung remain undilated. This is observed not infrequently in feeble and prematurely delivered children,

partly as the result of weakness of the respiratory muscles, partly from diminished irritability of the respiratory centre in the medulla. Abnormal conditions during labor (too rapid or prolonged labor, compression or torsion of the cord, narrow pelvis, forceps delivery, version, placenta prævia) favor the development of atelectasis. The bronchi may be occluded by inspired mucus or meconium, so that the corresponding portion of the lung remains atelectatic. Atelectasis is often associated with asphyxia neonatorum.

Obstruction or absorption atelectasis develops after occlusion of the bronchi, and gradual absorption of the air in the corresponding alveolar districts.

Lichtheim found, by experiments on animals, that the absorption of air in the lungs is effected by the blood circulating in the walls of the alveoli. Oxygen is first absorbed, then the carbonic acid, and, finally, the nitrogen.

Catarrh of the finer bronchi is the most frequent cause of obstruction atelectasis. It is therefore found with special frequency in the capillary bronchitis of childhood, either when it occurs as an independent disease, or secondarily to measles, whooping-cough, croup. It also follows obstruction of the bronchi by blood, fibrinous exudation, and foreign bodies. In rarer cases, it is the result of compression of a main bronchus by lymphatic gland or other tumors.

Compression atelectasis is produced by pressure upon the parenchyma of the lungs, for example, in pleurisy, pneumothorax, tumors of the pleura or lungs, pericarditis, dilatation or hypertrophy of the heart, tumors of the heart, aneurisms, mediastinal growths, spinal curvature associated with deformity of the thorax, meteorism, abdominal tumors, ascites, etc.

Marasmic atelectasis develops during the course of long-standing wasting diseases. It is most frequent in typhoid fever, and is not rare in other febrile diseases, and in the intestinal catarrh of children. Several factors often contribute to its development. In febrile diseases, these include parenchymatous inflammation and degeneration of the muscles of inspiration. If there is great general weakness, the irritability of the respiratory centre is diminished, and the interchange of gases in the lungs is not performed effectually. Furthermore, we must take into consideration the protracted retention of a certain position of the body, which occurs so commonly in very feeble individuals.

The great significance of an unchanged position of the body is recognized in that form of atelectasis known as physiological atelectasis. If the lower borders of the lungs of healthy individuals, who have slept on the back for some time, are auscultated in a sitting position, and, at the same time full inspirations are taken, we will very often hear a few fine râles during the first three to five inspirations. This is evidently explained by the fact that the lower posterior portions of the lungs take very little part in respiration while the individual is in a deep sleep and in the dorsal decubitus, so that the air partly disappears from the alveoli, and the alveolar walls come into apposition. When the alveoli are refilled with air by the deep inspirations, their walls are separated from one another, and the fine râles are thus produced.

II. ANATOMICAL CHANGES.—Atelectasis rarely extends over an entire lobe or lung—it is usually lobular. In congenital atelectasis, the base of the lungs is most frequently affected, then the anterior lower border and the lingual process of the left lung, rarely the apex. In



obstruction and marasmic atelectasis, the lower posterior portions of the lungs are affected most frequently. It sometimes appears as a stripe, about five cm. broad, extending upwards along the spine, and growing narrower towards the apex. The site of compression atelectasis depends upon the cause in each individual case.

Atelectatic foci are almost always situated at the periphery of the lung, rarely in the interior. Those following occlusion of the bronchi are generally wedged-shaped, with the broad base situated at the surface of the lung.

Atelectatic parts of the lung are depressed below the general surface. They are irregularly angular, following the peripheral boundaries of several lobuli. Their surface is usually uneven; their color may be brownish-red, bluish-red, or pale-gray (particularly in compression atelectasis). If the lungs are distended with air through a large bronchus, the depressed portions rise to the level of the remainder of the lung. They assume the normal color unless the parts have become congested. In such an event, they have a brick-red or cinnabar-red color.

The unaërated parts feel hard, do not crepitate, are flaccid but not brittle, occasionally leathery and tough (particularly in compression atelectasis). Pressure does not express air-bubbles, but at the most a serous or sero-bloody fluid. In obstruction atelectasis, mucous, purulent, or cheesy secretion may be squeezed out of the bronchi. Small pieces of atelectatic lung sink in water.

Atelectasis is often complicated with hyperæmia, so that the tissue acquires a bright-red color (carnification). Serous fluid may then accumulate in the alveoli, and the parts then look and feel like the splenic pulp (splenization). Finally, inflammatory changes develop in the alveoli. Bartels and Ziemssen showed that atelectasis often precedes pneumonic and broncho-pneumonic changes. Atelectatic spots are often surrounded by emphysematous portions.

Further changes occur even if the atelectasis is uncomplicated. After a time, the atelectatic parts can no longer be filled with air from the bronchus. Rokitsansky found fatty degeneration of the alveolar epithelium and proliferation of the interalveolar and interfundibular connective tissue. In the atelectatic portions of the lungs of children who died thirty-six to forty-eight hours after tracheotomy in croup, Balzer found dilatation of the interlobular and perilobular blood-vessels, infiltration of the tissue with a homogeneous, albuminoid exudation, filling of the alveoli with white blood-globules and desquamated epithelial cells, and particles of dust within and without the alveoli (probably aspirated through the tracheal canula). Balzer evidently had to deal, not with pure atelectasis, but with beginning broncho-pneumonia.

There are often changes in the circulatory channels. In congenital atelectasis, the foramen ovale and ductus Botalli are usually patent, and the right heart dilated. Gerhardt attributes thrombosis of the cerebral sinuses to circulatory disturbances in the domain of the right heart consequent on atelectasis. Thrombosis of the right heart has also been observed. The right heart is also dilated, as a general thing, in acquired atelectasis.

III. SYMPTOMS.—This condition produces disturbances in the pulmonary interchange of gases, unless the extent of the affected parts is so slight that their function may be assumed by adjacent parts.

The existence of congenital atelectasis should be expected if the children are apparently still-born. They breathe superficially, lie very

quiet, whimper very softly, do not take the breast, or soon cease nursing. If these symptoms increase in severity, the face becomes pale-gray and livid, the pulse very frequent, the sensorium clouded, and death from suffocation occurs, often preceded by twitchings or convulsions. The symptoms are those of increasing carbonic-acid poisoning.

Objective changes in the respiratory organs are found only when the atelectasis is widespread. We then notice inspiratory retraction of the chest, particularly in the upper and lower parts of the thorax. If the atelectatic portions attain a circumference of four to six cm., and a thickness of at least two cm., gentle percussion may give rise to dullness. Bronchial breathing and increased vocal fremitus, even increased bronchophony may be present.

The symptoms of acquired atelectasis are the same in kind as those of the congenital form. In addition, the development of the process is preceded by the occurrence of crepitant râles. It is especially characteristic of atelectasis, when dullness and crepitant râles disappear on taking full breaths, and changing the position of the body.

If the air in the atelectatic portions is merely diminished in amount and has not entirely disappeared, a tympanitic note will be produced on percussion (from diminished tension of the lung tissue).

Extensive atelectasis is usually followed by changes in the circulatory apparatus. Cardiac dullness is increased on the right side, and the diastolic pulmonary sound is intensified (signs of stasis in the pulmonary artery).

IV. DIAGNOSIS.—The recognition of pulmonary atelectasis is not easy, even if the process is extensive. The diagnosis depends on the demonstration of respiratory disturbances, associated with absence of air in the pulmonary parenchyma.

The disease is differentiated from pneumonia by the changes in the percussion sound on change of position and after deep inspirations. In addition, consonant râles are almost always heard in pneumonia.

In hemorrhagic infarction, we also find crepitant râles and perhaps dullness, but a bloody sputum is expectorated; and, in addition, the etiology differs.

Compression atelectasis is readily mistaken for pleurisy; but in the latter we usually find a linear boundary of the dullness, and, moreover, the vocal fremitus is diminished.

V. PROGNOSIS.—Apart from the extent of the atelectatic districts, the prognosis depends upon the causes of the disease. In congenital atelectasis, it is especially unfavorable if the infant is feeble and prematurely born, or if the "besoin de respirer" has been very much diminished by the pressure of a narrow pelvis, or of the forceps during delivery. Congenital atelectasis often persists for many weeks. Koestlin described a case in which congenital atelectasis of an entire lung was found in an autopsy on a young man of 22 years.

The prognosis of acquired atelectasis likewise depends upon the primary affection.

VI. TREATMENT.—The prophylaxis of acquired atelectasis includes the modern treatment of febrile affections which endeavors to prevent weakness of the respiratory muscles, and depressed irritability of the respiratory centre. Furthermore, the position of the body of very feeble patients should be changed hourly.

In congenital atelectasis, the mouth and air-passages should be cleared of mucus or meconium, and artificial respiration instituted.

The infant should be taken frequently from the bed and made to cry. Vigorous inspirations may be produced by placing the child in a warm bath, at the same time douching the chest with cold water. Stimulating baths of wine, calamus root (500. infused in 2 litres of water and added to the bath), and valerian are also indicated in feeble children.

In treating acquired atelectasis, we must first endeavor to remove its cause. The position of the patients must be changed frequently, and deep inspirations produced by warm baths and cold douches. Juergensen recommends the application of the douche to the occipital region over the medulla oblongata.

### 5. *Hypostasis of the Lungs.*

I. ANATOMICAL CHANGES.—Pulmonary hypostasis may develop whenever a certain position of the body is retained for a long time, and the vigor of the heart's action is diminished. Both these factors aid in the development of stasis of the circulation in the dependent parts of the lung.

The condition always begins with venous congestion and capillary stasis, the affected parts assuming a dark-blue to black-red color. The cut surface discharges, on pressure, a large amount of sticky, bloody fluid. Under the microscope, the vessels in the walls of the alveoli are found distended with blood, and sinuous in places. The slow circulation exercises a noxious influence on the walls of the vessels and makes them permeable. The venous hyperæmia is soon followed by transudation into the alveolar spaces. At the same time the alveolar epithelium swells and is partly desquamated. White and red blood-globules, which have passed through the walls of the vessels, are also mingled with the fluid contents of the alveoli. The microscopical appearances are very similar to those of catarrhal pneumonia and cannot always be differentiated anatomically. It differs genetically from catarrhal pneumonia, inasmuch as it is always the result of purely mechanical causes, and differs clinically in its apyrexial course. The lungs are now infiltrated with fluid to a greater extent than at the period of venous congestion; they are heavy and flaccid. This condition has been improperly termed hypostatic pneumonia.

Hypostatic changes are associated not infrequently with atelectasis and pulmonary œdema. Pneumonic changes also occur as secondary processes under certain conditions.

The posterior inferior parts of the lungs are affected most frequently; more rarely it is observed only on one side.

II. ETIOLOGY.—The most frequent causes of the prolonged retention of one position of the body and of weakness of the heart are febrile infectious diseases, particularly typhoid fever. Hypostasis may also follow joint diseases, paralysis, and fracture of the bones, if the patients are compelled to retain a certain position of the body, or are enfeebled from other causes, especially from old age. Furthermore, pulmonary hypostasis also develops not infrequently during a prolonged death-struggle in any disease.

We must also call attention to the fact that the occurrence of pulmonary hypostasis is favored by certain auxiliary factors, which impair the respiratory movements, and thus affect the pulmonary circulation (pleuritic adhesions, spinal and thoracic deformities, elevated position of the diaphragm as the result of tympanites, ascites, or abdominal tumors).



It must also be remembered that the weight of the body impedes respiration when the body is kept in one position.

III. SYMPTOMS.—The symptoms of pulmonary hypostasis are readily overlooked during life, and the objective disturbances may be very slight.

Attention is often attracted by accelerated breathing, cyanosis, and increasing apathy (carbonic acid narcosis). Cough may be absent; in other cases the patients expectorate a mucous, muco-purulent, or even bloody sputum. The temperature of the body is unaffected, so long as inflammatory complications remains absent.

The percussion sound is slightly dull and tympanitic over the hypostatic portions of lung. The respiratory murmur is feeble, and fine, non-consonant râles are heard. If the fluid in the alveoli has increased to such an extent as to exclude the air over a considerable area, the physical signs of inflammation of the lung will make their appearance, viz.: dullness, bronchial breathing, consonant râles, increased vocal fremitus, and bronchophony. In such cases, hypostasis and pneumonia cannot be differentiated clinically, particularly if the case has not been followed from the beginning, and if the primary affection has given rise to febrile movement.

Pulmonary hypostasis may develop within a few hours and disappear with equal rapidity. It sometimes lasts for many days or even for weeks.

IV. DIAGNOSIS.—Pulmonary atelectasis is distinguished from hypostasis by the fact that the former disappears more rapidly after changing the position of the body, but a positive differential diagnosis cannot be made in all cases.

In pneumonia, the phenomena of consonance predominate, as mentioned above, but here also the diagnosis may present insoluble difficulties.

In œdema of the lungs, dullness is rare, and the râles are diffused more widely over the lungs.

V. PROGNOSIS.—This is grave on account of the etiology of the affection. The weakness of the heart is apt to become excessive, or the hypostasis is followed by secondary inflammation of the lungs, which proves fatal.

VI. TREATMENT.—The position of apathetic patients or those who are motionless in bed from other reasons, should be changed every two hours. Heart-failure is best prevented in febrile conditions by the use of antipyretics and baths, and by large doses of alcohol. Stimulants are sometimes indicated, for example,  $\mathcal{R}$  Acid. benzoic., 0.3; camphor. tritæ, 0.05; sacch. alb., 0.5. *M. f. p., d. t. d. No. x.* *S.* One powder every two hours.  $\mathcal{R}$  Liq. ammon. anisat., 10.0; gtt. v. every one to two hours.  $\mathcal{R}$  Moschi, 0.1; sacch. alb., 0.5. *M. f. p.* To be taken every hour.

## 6. *Œdema of the Lungs.*

I. ETIOLOGY.—Pulmonary œdema is the term applied to filling of the alveoli with serous transudation. It is associated with œdematous infiltration of the interstitial tissue. According to some writers, the latter condition occurs occasionally as an independent disease.

The development and duration of pulmonary œdema may last either several hours or several days (acute and chronic œdema). The œdema

may be confined to small portions of the lung, or it may extend over an entire lung and even to both lungs.

Œdema of the lungs rarely occurs as an acute idiopathic and independent disease. This has been observed after the ingestion of cold drinks, drenching with cold water, and catching cold from sudden changes in temperature. In one case, Struempell was unable to find any cause. It is doubtful whether, under such circumstances, we do not have to deal with inflammatory and not with purely transudative changes; in other words, we suspect that the condition in question is a rare form of serous pneumonia in which a coagulable exudation is not deposited within the alveoli.

There is no doubt of the inflammatory character of that form of circumscribed œdema which is found in the immediate vicinity of inflammatory foci in the lungs (croupous pneumonia, abscess, neoplasm, etc.). It constitutes, to a certain extent, the prolongations of the inflammatory process, in which exudation of fluid from the vessels still takes place, but migration of white blood-globules does not occur.

It has been held that arterial congestion of the lungs sometimes gives rise to œdema, but this view has been properly contradicted. The occurrence of collateral œdema of the lungs is also more than doubtful.

In the large majority of cases, œdema of the lungs is a complication of other diseases, and is the result of stasis in by far the largest proportion of cases. Cohnheim and Welch proved experimentally that paralysis of the left ventricle, which offers insurmountable obstacles to the circulation of blood from the intact right heart, always furnishes the condition necessary to the development of pulmonary œdema. Such stasis œdema is extremely apt to develop during the death struggle (agonal pulmonary œdema), when the left heart has ceased to contract while the right ventricle is still acting. Its development is also favored by changes in the heart muscle, whether the result of valvular lesions, diseases of the pericardium or coronary arteries and renal diseases, or of fever and infection. Diseases of the central nervous system may also give rise to œdema of the lungs by interfering with the heart's action.

Stasis œdema also includes the hypostatic form observed in feeble individuals, who have retained the same position of the body for a long time. If the patients have remained constantly on one side, the œdema generally involves the corresponding lung; in protracted dorsal decubitus, the œdema is bilateral.

Pulmonary œdema is sometimes the result of morbidly increased permeability of the blood-vessels, as was proven experimentally by Cohnheim and Lichtheim, in animals whose blood was rendered poorer in albumin. In man, this is observed in many cachectic conditions (Bright's disease, cancer, phthisis, etc.).

Pulmonary œdema sometimes occurs as a sort of œdema ex vacuo, the alveoli being in the same condition as the skin during the process of capping. This category includes the variety which develops in stenosis of the air passages by foreign bodies, fibrinous exudation, etc. We are also inclined to include in this class the œdema which follows the inhalation of irrespirable gases, for example, carbonic oxide, although some authors attribute it to increased permeability of the walls of the vessels.

II. ANATOMICAL CHANGES.—If the œdema is general, the volume of the lungs is increased; they appear swollen, and collapse very little or

not at all. They have a peculiar spongy feel, and the pressure of the finger leaves a more or less deep groove, which disappears more rapidly in acute than in chronic œdema. The lungs are often extremely pale and have an almost transparent appearance.

The cut section discharges a profuse, thin, finely frothy fluid, with which not alone the alveoli, but also the bronchi are partly filled. The fluid is sometimes watery, sometimes of a rosy color, sometimes mixed with considerable blood. In jaundiced bodies it may have a yellowish color; in cases of heart disease and brown induration of the lungs it has a brownish tinge, and in deeply pigmented lungs it is blackish.

The frothy character of the fluid is an important point in the anatomical diagnosis, because fluid not infrequently passes into the alveoli after death, but is naturally destitute of air.

As a result of œdema, the tissue of the lung sometimes becomes very brittle.

III. SYMPTOMS.—The result of œdema of the lungs is a disturbance of the pulmonary interchange of gases which may increase to suffocation.

Respiration is accelerated and difficult as under other similar conditions. The signs of cyanosis become visible upon the skin and mucous membranes. If the carbonic-acid poisoning becomes excessive, the sensorium becomes clouded, the face assumes a leaden gray color, somnolence is produced. Twitchings occur in individual muscles or in groups of muscles, and finally death from suffocation.

As a rule, expectoration becomes very profuse with the development of this affection. The sputum is thin, frothy, watery, of a light yellow color, or rosy from the admixture of red blood-globules, more rarely mingled with streaks of blood. It contains albumin, a small amount of mucin, a few pus-corpuscles, red blood-globules, and swollen alveolar epithelium cells.

Fleischer found a not inconsiderable quantity of urea in the sputum of a patient who was also suffering from renal disease.

As a rule, the profuse expectoration does not continue long, as the muscles grow weak, and the secretion remains in the air passages. This evidently increases the danger. At the same time a peculiar rattling is heard in the chest. Coarse râles are also formed in the trachea, and are often the sign of impending dissolution.

On auscultation, fine crepitant râles are heard, which are produced during inspiration by the tearing of the alveolar walls from the fluid. But as the bronchi are almost always filled with fluid, we usually find widespread and extremely profuse moist râles (coarse, medium-sized, and fine). The râles are clear, but not consonant, and usually conceal the respiratory murmur.

Percussion either reveals no change, or the percussion sound is lower than normal and slightly tympanitic.

If the fluid in the alveoli loses all the air contained in it, dulness, bronchial breathing, and increased vocal fremitus may be produced.

IV. DIAGNOSIS.—The recognition of pulmonary œdema is usually easy if the affection is not very circumscribed. The diagnosis is rendered positive by the profuse expectoration of a chiefly serous sputum with the signs, described above, of a collection of fluid within the pulmonary alveoli.

V. PROGNOSIS.—The prognosis is always very serious. Œdema of the



Lungs in itself is a dangerous disease, and in addition the causes of the disease are often of such a grave nature that not much can be expected from treatment. If death occurs in a short time—almost in an apoplectic form manner—the condition has been termed serous apoplexy of the lungs.

VI. TREATMENT.—Great attention should be paid to prophylaxis. The vigor of the heart's action should be maintained in all febrile diseases, and this is best done by the modern treatment of fever. In Bright's disease and other affections which are associated with loss of vital fluids, this loss should be diminished as much as possible. In very feeble individuals the position of the body should be frequently changed, in order to prevent hypostasis.

If pulmonary œdema has developed, the main indication is to increase the vigor of the heart. Wine and stimulants should be given copiously.

If the œdema is far advanced, we should also endeavor to keep the air passages free. Expectorants, combined, if possible, with stimulants, are advisable (acid. benzoic., 0.3; camphor. trita., 0.05; every two hours. Decoct. rad. seneg., 10.0:180; liq. ammon. anisat., 5.0; syr. simpl., 15.0; one tablespoonful every two hours, may be administered). The advisability of administering emetics is doubtful, as they increase the loss of power, and often prove ineffective in advanced carbonic-acid poisoning.

Derivation to the skin is often employed (alcoholic inunctions, mustard poultices, mustard hand and foot baths, fly blisters, dry cups, etc.). Traube recommended acetate of lead internally.

In œdema of the lungs owing to Bright's disease, diaphoretics, diuretics, and drastics have been largely employed without any special benefit. In such cases the plan of treatment recommended above is most effective.

Venesection is an important feature in treatment. In many cases, however, its effects are only temporary, and it would be entirely wrong to omit, on account of this measure, the stimulation of the heart's action.

### 7. *Catarrhal Inflammation of the Lungs.*

#### *(Broncho-pneumonia. Lobular Pneumonia.)*

I. ETIOLOGY.—In catarrhal pneumonia the alveolar spaces are filled with fluid exudation. The disease is always secondary, and is preceded by catarrh of the finer bronchi. It always appears—at least in the first stages—in small, scattered foci, and hence the name lobular pneumonia.

The disease develops chiefly in children and old people, rarely in vigorous individuals at the prime of life. Sex appears to exert no influence.

In children the disease is especially frequent in the second and third years of life, rare in the first six months. This is partly owing to the fact that better care is taken of the younger infants, and also because they are free from the infectious diseases which give rise not infrequently to broncho-pneumonia.

It occurs most frequently in the course of certain infectious diseases. Whooping-cough and measles occupy the first rank, then follow diphtheria of the pharynx or larynx, influenza, r  theln, scarlatina, small-pox, typhoid fever, dysentery, erysipelas, and miliary tuberculosis. The complication with broncho-pneumonia depends upon the

character of the epidemic rather than upon accidental noxious influences to which the patients were subjected.

Catarrhal pneumonia is sometimes the result of bronchitis, the danger of this complication being so much greater the finer the bronchi which are affected. As a matter of course, the causes of the broncho-pneumonia are then the same as those of bronchitis (vide page 203).

Feeble, anemic, scrofulous, and rachitic children are most liable to it, likewise those who live in dusty, poorly-lighted, crowded apartments. Bronchitis and broncho-pneumonia develop not infrequently during dentition. The number of cases increases during cold and changeable weather (January to April).

Foreign bodies in the finer air passages may give rise to capillary bronchitis, and then to broncho-pneumonia. A single attack leaves behind a tendency to relapse.

Different views are entertained concerning the relation of capillary bronchitis to broncho-pneumonia. Some writers assume a direct propagation of the inflammation from the bronchial mucous membrane to the walls of the alveoli. v. Buhl believes that inflammatory products are aspirated into the alveolar spaces from the finest bronchioles, and then produce secondary inflammation. By others, atelectasis was assumed as the connecting link between capillary bronchitis and broncho-pneumonia. According to recent views, however, inflammation occurs only when inflammation-producers have been present. Atelectasis in itself does not give rise to inflammation, but it favors, perhaps, the spread and development of the inflammation-producers.

The nature of the inflammation-producers is still in doubt. In our opinion they consist of bacteria, so that catarrhal pneumonia should really be included among the infectious diseases. Buhl, Wyss, and others have demonstrated colonies of bacteria in broncho-pneumonic spots. The same bacterium has not been found in all cases. On the contrary, the disease seems to be produced by very different schizomycetes, which are in part specific of the individual infectious diseases. We are also inclined to think that, in "foreign-body pneumonia," the foreign body does not act *per se*, but rather in its capacity as carrier of bacteria. We would call attention to the fact that this form of the disease is not infrequent in infectious diseases, as the result of the entrance of buccal secretion and débris of food into the air passages of apathetic and feeble patients. It has been acknowledged that a cold is not without influence upon the development of pneumonia, but it should not be regarded as a directly noxious influence, but rather as one which favors the entrance of bacteria.

II. ANATOMICAL CHANGES.—Catarrhal pneumonia occurs almost always in circumscribed spots, the number and size of which are liable to great variations. They vary in number from a few to more than a hundred, and in size from that of a pin's head to that of a walnut. If the foci are in close proximity they may finally coalesce, so that the originally lobular disease is converted into a lobar disease.

As a rule, the foci are situated peripherally, extending close under the pulmonary pleura. They are found exclusively, or at least in greatest numbers, in the inferior posterior portions of the lungs. They sometimes form a continuous streak which begins at the base of the lung next to the spinal column and, growing gradually smaller, extends along the spine towards the apex. The spots of inflammation are very often bilateral.

The lesion is easily recognized. On palpation from the outside, the lung appears infiltrated with firm nodules. Upon section these present a bluish-red or brownish-red color, are entirely empty of air, have a uniform smooth, shining surface, and sink in water. Air cannot be blown into the foci through the bronchi.

The bronchi which enter the foci contain mucous, puriform, or slightly thickened secretion, which can be expressed not infrequently from the bronchi in the shape of a sausage. The bronchial mucous membrane is generally reddened, loosened, swollen, occasionally ecchymotic. The lumen of the bronchus is sometimes dilated cylindrically.

In addition to the broncho-pneumonia, we often find atelectatic and acutely dilated spots. The dilatation affects chiefly the anterior median borders and the anterior surface of the upper lobes.

The pleura and bronchial glands almost always take part in the inflammation. Upon the pulmonary pleura are found ecchymoses and membranes; serous and purulent effusions into the pleural cavity are very rare. The bronchial glands are swollen, succulent, and hyperæmic.

Traube showed that the pulmonary changes which follow section of both pneumogastrics in animals are the result of catarrhal inflammation. Friedlaender found that this change begins with serous infiltration of the pulmonary alveoli, in consequence of which the alveolar epithelium is swollen and loosened. Then follow inflammatory changes: dilatation of the blood-vessels, emigration of white blood-globules, and their entrance into the interstitial connective tissue and the alveolar spaces. According to Dreschfeld, the nuclei of the alveolar epithelium undergo proliferation from the beginning, so that the epithelium plays an active part and the process begins with inflammatory changes. Juergensen and Schueppel produced catarrhal pneumonia in rabbits by causing them to inhale irritating gases (chlorine, ammonia). Similar changes may be produced in man by the inhalation of the vapor of irritating acids.

In the catarrhal pneumonia of the human lung, the alveolar spaces are found filled with swollen alveolar epithelium cells, which are partly granular and fatty, and with white and a few red blood-globules; the pulmonary capillaries are dilated and sinuous, and the interstitial tissue is infiltrated with round cells. In a case of resolving inflammation, Cornil observed that the elastic fibres in the interstitial tissue were unusually broad, swollen, and refractile, presented irregular transverse projections, and broke readily into small sections (Schwalbe noticed similar appearances in the elastic fibres of the ligamentum nuchæ; these were produced artificially by maceration in chromic acid).

III. SYMPTOMS.—As a rule, the clinical history of catarrhal pneumonia is very vague. This is easily understood if we remember that the process is usually a complication of other diseases, particularly the infectious diseases, which attract the chief attention. Moreover, the changes in the lungs produced by the catarrhal inflammation are often very slight, and though it may not be difficult to recognize an existing bronchitis, the complicating pneumonia may be overlooked by the most careful diagnostician.

Broncho-pneumonia may be either acute or subacute, the former running a febrile course in one to four weeks, the latter presenting remissions and exacerbations and lasting much longer. The pneumonia of measles is usually acute, that of whooping-cough is subacute.

According to Ziemssen, the temperature in simple bronchial catarrh



rarely exceeds  $39.0^{\circ}\text{C}$ . so that higher temperatures which last for several days favor the view of a complication with catarrhal pneumonia. The fever presents no distinct type. As a rule it is remittent, the lower temperature appearing usually in the morning. Defervescence occurs gradually (vide Fig. 70).

The frequency of the pulse increases with the temperature of the body and, in children, not infrequently passes beyond two hundred beats in a minute. The respirations are also increased in frequency and occasionally number nearly one hundred in a minute (combination of febrile influences and of disturbance of respiration by the inflamed lung). An important feature is the production of pain on coughing and breathing, since this is rarely observed in simple bronchitis. The little patients

FIG. 70.



Temperature curve in catarrhal pneumonia in a girl æt. 4 years.

cry on coughing or distort the features; palpation of the thorax also produces pain. The respiratory movements are irregular and jerky, expiration is often groaning or moaning, and speech is likewise short and interrupted. These symptoms must be attributed to irritation of the pleura, and to the pain produced thereby by incautious respiratory movements.

Local changes in the lungs are often absent, or are very insignificant, and admit of various interpretations. There are frequently signs of objective dyspnoea (movement of the alae nasi, inspiratory retraction of the intercostal spaces, inspiratory depression of the lower costal cartilages and the epigastrium, etc.), but these do not constitute a specific symptom. Increased vocal fremitus may be expected only when larger parts of the lung have been deprived of air, and form lobar foci of disease; but it should not be forgotten that bronchitis may present temporarily the conditions for increase of vocal fremitus (occlusion of the bronchi by secretion). Dulness may be expected over peripheral foci which are more than five cm. in circumference and two cm. in thickness. The dulness is usually bilateral, and extends in a stripe along the spinal column. Distinct dulness is often absent, but there may be a tympanitic percussion sound, which is the result of relaxation of the pulmonary tissue, in consequence of occlusion of the finer air passages, and beginning collapse of the lungs. An increased feeling of resistance should be carefully looked for on palpatory percussion.

The auscultatory signs demand an experienced ear. When other

methods of examination give negative results, pneumonia may often be recognized by the clearness and consonance of the râles. Over larger spots of disease, we find increased bronchophony, which is especially distinct during the expiratory groaning or crying of the little patient. Finally, bronchial breathing is heard over large infiltrations.

As a rule, no sputum will be observed, because it is generally swallowed by children and old people. In other cases it is muco-purulent, occasionally streaked with blood.

If the disease ends in recovery, the fever subsides, the appetite reappears, and the local changes gradually diminish. Under certain circumstances, several weeks may elapse before this takes place. Sometimes pneumonic foci may disappear while others are forming in other places (subacute form). Death may result from increasing emaciation and exhaustion, or from heart failure if the temperature is very high, or finally from carbonic-acid poisoning and suffocation. In the latter event, the skin grows pale, the mucous membrane livid and leaden gray. The sensorium is obtunded, twitchings occur in the face or limbs; Cheyne-Stokes respiration becomes noticeable, and finally the respirations cease.

In subsequent sections we will discuss the terminations in phthisis, miliary tuberculosis, abscess, gangrene, interstitial connective-tissue proliferation, and retraction of the lungs.

Complications sometimes arise. Herpes, sudamina, purpura, ecthyma, furuncles, and gangrene have been observed upon the skin. Noma has also been mentioned. Attacks of laryngeal dyspnoea sometimes develop, and are attributable to catarrhal swelling of the laryngeal mucous membrane. Croup and diphtheria may occur secondarily. Slight albuminuria is usually of a febrile character, but Minot reports cases of parenchymatous nephritis and attacks of hæmoglobinuria. Pleurisy with effusion and pericarditis have been observed occasionally, endocarditis and meningitis more rarely. Pneumothorax, interstitial and subcutaneous emphysema have been described, also epistaxis and otitis. The fatal termination is sometimes accelerated by obstinate diarrhoea and vomiting.

IV. DIAGNOSIS.—A probable diagnosis can alone be made in many cases of catarrhal pneumonia. If infiltration of the lungs is demonstrable, we must differentiate it from croupous pneumonia, phthisis, and atelectasis.

It is distinguished from fibrinous pneumonia by its occurrence on both sides, the demonstration of multiple small foci, the absence of a cyclical course and of the rusty sputum. The differentiation from pulmonary phthisis is effected by an examination of the sputum for tubercle bacilli. In atelectasis, the symptoms are not infrequently of a temporary character, and may often be made to disappear by changing the position of the patient and securing deep inspiration.

V. PROGNOSIS.—The prognosis is always grave. In crowded hospitals for infants the mortality is sometimes frightful.

Valleix lost 127 patients out of 128; Bouchut, 22 out of 55. Even under favorable circumstances, the mortality averages from thirty-three to thirty-six per cent.

The factors to be taken into consideration in prognosis are : Extent of the inflammatory changes, height of the fever, constitution, age, and primary disease. In the first six months of life—in which the disease is rare—the mortality is very high. As a general thing, the pneumonias

following pertussis and small-pox are graver than those following measles.

Among the unfavorable individual symptoms are signs of carbonic-acid poisoning, Cheyne-Stokes respiration, and great frequency of the respirations (50-70 per minute) in apyrexial conditions when the local symptoms are unchanged.

VI. TREATMENT.—The prophylaxis is in great part the same as that of bronchitis. If catarrhal pneumonia has developed, the patient should be placed in a roomy, bright apartment, which can be aired through the adjacent room. The temperature of the room should be kept constantly at 15° R. The air should be kept moist by placing vessels of water on the stove, inhalation apparatus or spray. The patients should be bathed morning and evening (26° R.) for fifteen to twenty minutes. In addition, good beef-tea, milk, eggs, and wine ( $\frac{1}{2}$ -1 tablespoonful every hour) should be given. If the fever is high, we may give antipyrin (1.0-2.0 to 50.0 of lukewarm water as an enema). Less certain effects are obtained from quinine, salicylic acid, benzoate of soda, kairin. Warm poultices are serviceable in severe pains in the chest. If numerous râles are heard, we may give expectorants, under certain circumstances emetics (vide page 219). If signs of carbonic-acid narcosis develop, we should give a warm bath, and apply a stream of cold water to a point readily found immediately beneath the occiput, and from which, as Juergensen showed, deep, almost dyspnoeal inspirations may be produced. Cold douches to the anterior chest-wall, while the patient is in the warm bath, also act in a similar manner. The children should be carefully watched during convalescence, and later should be sent to the country.

### 8. *Fibrinous Inflammation of the Lung.*

(*Fibrinous Pneumonia. Croupous Pneumonia*).

I. ETIOLOGY.—Fibrinous pneumonia leads to the formation of a coagulable exudation, rich in fibrin, which fills the alveolar spaces and drives out the air. It always appears in large foci, so that extensive portions of a lobe, an entire lobe or lung is affected (lobar pneumonia). The inflammation is rarely confined to the alveoli, but the finer bronchi are almost always attacked by the fibrinous inflammation, and in rare cases this extends even to the larger bronchi.

The term croupous pneumonia is used by many physicians instead of fibrinous pneumonia. We believe, with Virchow, that the former term should be applied only to those inflammations of the lung which develop as the result of laryngeal croup.

A strict etiological distinction should be made between primary and secondary fibrinous pneumonia.

The secondary form develops occasionally in the course of infectious diseases, for example, typhoid fever, small-pox, scarlatina, measles, etc. It is sometimes the result of malarial infection and runs an intermittent course.

Secondary pneumonia also occurs in protracted, exhausting diseases, not infrequently as a terminal condition, for example in Bright's disease, diabetes, cancer, etc.

Primary fibrinous pneumonia occurs as an independent disease.

The views concerning the nature of primary pneumonia vary more



than ever at the present time. In our opinion it is an infectious disease, whether it develops primarily or secondarily. Catching cold possesses a causal significance only in so far as it favors the occurrence of infection.

The following are the arguments advanced in favor of the infectious character of fibrinous pneumonia: *a.* The disease almost always runs a typical, cyclical course—a characteristic which is especially peculiar to infectious diseases. *b.* The general symptoms are independent of the local changes in the lungs, inasmuch as the fever and other signs of general infection precede the local changes and usually disappear before the latter. This would be inexplicable if the disease were a local affection produced by a cold. Moreover, there is not infrequently a great disproportion between the local and general changes. *c.* The disease ordinarily occurs epidemically and endemically. It may be said that the agencies which produce colds may also affect a large complex of people and thus lead to an epidemic outbreak, but not a few epidemics of pneumonia have been known to occur during the most favorable meteorological conditions. Furthermore, those individuals who work in the open air, and are most exposed to the inclemency of the weather, by no means furnish the principal proportion of cases of pneumonia. *d.* After Klebs had found bacteria in the bronchial secretion of fibrinous pneumonia, similar discoveries were made by other observers. The most exhaustive researches were made by Friedländer. Under the name pneumococci he describes ellipsoidal bodies, with a length of about  $1.0\ \mu$  ( $1\ \mu = 0.001\text{ mm.}$ ), and a breadth equal to one-third the length. They are arranged usually in groups of two or more, and are surmounted by a clear gelatinous envelope. Friedländer obtained pure cultures, which assumed macroscopically the shape of a nail, and gave rise to fibrinous pneumonia on being inoculated in animals. But A. Fraenkel claims that the gelatinous envelope is not characteristic of pneumococci, inasmuch as they also occur in other bacteria, for example, those found in the buccal cavity of healthy individuals, and on the other hand are absent occasionally in pneumococci. Moreover the pneumococci have not been studied sufficiently in all directions.

The infectious nature of fibrinous pneumonia being granted, it remains to decide whether it may be produced by one agent alone or by various forms of bacilli. We believe in the multiformity of the pneumonia poison, and when secondary fibrinous pneumonia develops in the course of certain infectious diseases, we think that certain bacilli which lie at the basis of such diseases are deposited in the pulmonary alveoli, here produce metastases, as it were, and give rise to inflammation. Indeed, we do not consider it absurd to believe that an agent which usually gives rise to another infectious disease may be deposited, contrary to the general rule, in the pulmonary alveoli and there produce fibrinous inflammation. From this standpoint we can explain the occurrence of pneumonia in typhoid fever, *i. e.*, a fibrinous pneumonia produced by the schizomycetes of typhoid fever, which have not been deposited, as is generally the case, in the intestines, but in the alveolar spaces of the lungs. On the other hand, the pneumococci may enter the general circulation and pass to more or less distant organs, producing metastasis and inflammation, for example, pericarditis, meningitis, nephritis (observations by Salvioli, Eberth, and Nauwerck).

The pneumococcus is probably received directly through the air passages. Some authors believe that there are cases of infection in which

the pneumococci are located only in the blood and produce general disturbances alone, while local changes in the lungs do not develop.

Primary fibrinous pneumonia is a very frequent disease. According to Holmsen, about 4 per cent of the population of Norway were treated, from 1869-1878, for fibrinous pneumonia. According to the statistics of four Russian hospitals collected by Bary, 3.8 per cent of the patients suffered from pneumonia.

Holmsen showed that the frequency of the disease varies greatly in different years. In Norway it was especially frequent from 1874-1876, so that eleven per cent of the population of Christiania and adjacent parts suffered from the disease. It was noticed at the same time that the disease gradually spread over larger tracts of the country.

The seasons exert a great influence on the development of the disease. The majority of cases occur from March to May; the disease is observed somewhat less frequently from December to February, while it is hardly half so frequent in the summer and autumn.

Pneumonia epidemics sometimes extend over a large town, or even a large section of country; in some cases, we have house epidemics. The duration of the epidemic may be many weeks, or even several months. Epidemics of other infectious diseases, particularly typhoid fever, diphtheria, and occasionally meningitis, often prevail at the same time. It is especially noteworthy that in the same way as there are typhoid fever houses, so there are also pneumonia houses, in which new cases of fibrinous pneumonia continue to develop for years. Certain observations seem to show that the vitality of the pneumococcus is very great, and Flindt states that it is retained for three years. In 1880, Kerschensteiner found that one hundred and sixty-one cases (fourteen per cent) of pneumonia developed among eleven hundred and fifty persons in the Amberg prison. In this case, Kellar discovered pneumococci in the ceiling of the dormitory in which most of the cases occurred, obtained pure cultures, and successfully inoculated animals.

Sporadic cases of the disease also occur and are generally admitted into hospitals, so that, while general practitioners, particularly country physicians, have long believed in the infectious nature of fibrinous pneumonia, this view has gained ground very slowly among hospital physicians and clinicians.

Direct causes of the outbreak of a pneumonia epidemic may sometimes be demonstrated. It has been known to develop in prisons, barracks, and hospitals, after overcrowding, bad ventilation of living and sleeping-apartments, uncleanness, and improper food. The digging of sewers, etc., in the neighborhood of dwellings has been repeatedly mentioned as the cause of an epidemic.

Every one who remains for some time in the infected locality is in danger of infection. Not that the pneumococci may develop from any decomposing mass, but that they find in such a locality favorable conditions for propagation. Furthermore, numerous observations favor the view that individuals who remain healthy may convey the disease from the infected spot to a healthy locality, and may there give rise to pneumonia.

A number of epidemics have been known to cease when, for example, troops were conveyed from unhealthy barracks to a healthy one, or when prisons were in part emptied of their inmates, and were kept in a more cleanly condition. In other cases, epidemics disappeared after heavy

rains, perhaps because the air and the surface of the soil, which were laden with bacilli, were thereby cleansed.

The attempt has been made to bring pneumonia epidemics into connection with the soil water, but this theory is being abandoned more and more.

As a rule, the causes of sporadic cases cannot be demonstrated. Catching cold is often adduced as a cause, but, on close questioning, it is usually found that the patients assert more than they are able to prove. Nevertheless, we believe that a cold exerts an influence in certain rare cases, but merely by acting as an auxiliary factor in infection.

Injury is sometimes mentioned as a cause of the disease. Among three hundred and twenty cases collected by Litten, fourteen (4.5 per cent) were said to be connected with traumatism. Whether this contusion-pneumonia is anatomically identical with genuine fibrinous pneumonia is not positively settled, but at all events it differs from it clinically in the frequent occurrence of a bloody sputum.

It has also been maintained that foreign bodies in the air passages may give rise to fibrinous pneumonia, but this has been disputed. Inflammatory changes always remain absent when wax balls are shoved into the bronchi. Nor has the development of fibrinous pneumonia from chemical irritants been proven.

Different views are entertained with regard to the duration of the period of incubation, *i. e.*, the period which elapses between the entrance of the bacteria into the human body and the first gross disturbances. In our opinion, this period is very short, often lasting only a few hours. In forming this opinion, we rely upon those cases in which a severe exposure was followed almost immediately by pneumonia. According to Flindt, the period of incubation averages two days, while, according to other authors, it varies from three to twenty-four days.

Experience teaches that genuine fibrinous pneumonia is more frequent in males than in females (among Bary's three thousand two hundred and seventy-two cases, two thousand five hundred and fifty-five were males, and seven-hundred and seventeen females). According to Schramm, this relation is reversed in old age.

The disease occurs at every age, but is more frequent beyond the age of fifteen.

It was erroneously held, in former years, that fibrinous pneumonia does not occur in children under five years. On the contrary, the disease is observed not infrequently during infancy.

Feeble individuals, or those exhausted by previous illness, old people, and those addicted to drink, manifest a marked predisposition to fibrinous pneumonia.

Repeated attacks of the disease are often observed, so that a single attack predisposes to subsequent inflammations. One of Andral's patients was attacked by fibrinous pneumonia sixteen times in eleven years, and Busch reports a case in which the disease appeared twenty-eight times within a few years.

II. ANATOMICAL CHANGES.—Fibrinous pneumonia is divided into three stages, *viz.*, engorgement, hepatization, and resolution. In almost all cases, however, we find, upon autopsy, that all these stages are present, evidently because the inflammatory process neither develops at the same time in all places nor does it always run an equally rapid



course. As a rule, we can only speak of the predominance of this or that stage.

In the stage of engorgement, the amount of blood in the affected parts of the lung is increased. They are intensely red and increased in size, and a depression remains behind after pressure with the finger. They crepitate very slightly or not at all on section. On pressing upon the cut section, a bloody, sticky, viscid fluid is discharged, which at first contains air bubbles, but later is destitute of air. Small sections of the lung will float in water so long as the fluid contains air bubbles in abundance; they sink as soon as the change in question reaches its height and the expressed fluid no longer contains air bubbles.

In the stage of hepatization, the inflamed lung is converted into a firm, non-aërated mass. The lung tissue is not readily torn, but it is brittle and sinks entirely under water. The cut section has a granular appearance, this appearance being most distinct when a side light is allowed to fall upon it. The granulation is much finer in children than in adults, and is especially coarse in old people and emphysematous individuals. This is owing to the fact that the individual granulations are fibrinous casts of the alveoli, and therefore correspond to the size of the latter.

The stage of hepatization includes various sub-stages, which follow one another chronologically as red, gray, and yellow hepatization. In red hepatization, the granular section of the lung has a red or reddish-brown color, the vessels and bronchi alone being visible as whitish streaks. The red color gradually gives place to a grayish-red and finally gray color (gray hepatization). In certain places the black pigment of the lung appears with extreme distinctness, especially in adults and old people. A section of the lung then presents a marbled appearance. The gray color of the diseased lung finally becomes grayish-yellow or yellow, pus-like in color (yellow hepatization).

In the stage of resolution, the granular appearance of the lung is rapidly lost. The lung is infiltrated with a fatty, yellow, pus-like fluid, which may be squeezed out in abundance from the cut surface (stage of purulent infiltration). If a piece of the lung is carefully washed in water and the contents of the alveoli removed, the ordinary spongy texture of the lung is readily recognized. But this must be done very cautiously, as the pulmonary tissue is extremely brittle and readily torn.

A part of the fluid exudation is removed by expectoration, the larger part is absorbed by the lymphatics. An affection of the lymphatic channels in the lung might lead, therefore, to serious disturbances of absorption. When the alveoli have been freed of the exudation, regeneration of the alveolar epithelium takes place immediately, and the *restitutio ad integrum* is complete.

The following are the microscopical changes in fibrinous pneumonia:

In the stage of engorgement, the vessels of the alveolar walls are dilated, congested, and sinuous in part, so that they project into the alveoli and narrow them. The alveolar epithelium cells swell up and desquamate in part. The epithelium cells which contain protoplasm undergo nuclear proliferation. A viscid fluid, rich in albumin, exudes from the dilated vessels, and emigration of white and red blood-globules takes place into the alveolar spaces. Extravasations of blood are occasionally met with.

The stage of hepatization is characterized by coagulation of the inflammatory product in the alveolar spaces. Granular and fibrillated

structures appear more and more among the cellular contents of the alveoli and inclose the cells between them. According to Veraguth, the flat alveolar epithelium cells take part in the coagulation, inasmuch as they degenerate into a granular fibrillated mass. Feuerstack does not confirm this statement; he noticed that the non-nucleated plates undergo fatty degeneration, and denies the implication of the alveolar epithelium in the process of coagulation.

At the period of red hepatization, the hyperæmia of the blood-vessels persists, and at the same time they may be artificially injected. The hyperæmia gradually subsides, and gray hepatization develops. The white blood-globules then emigrate actively, and in addition, according to Axel Key, the cells already present in the alveolar spaces undergo proliferation. When fatty degeneration of the cellular elements occurs, the stage of yellow hepatization is produced.

The stage of resolution begins with liquefaction of the coagulated masses, and at the same time the fatty degeneration of the cellular products increases more and more. According to Feuerstack, the regeneration of the alveolar epithelium is the result of a great increase in the number of the nucleated epithelium, these furnishing the germ of a new epithelium layer. According to other writers, the epithelium develops from emigrated round cells.

According to recent investigations, pneumococci form the most important constituent and are found, not only in the contents of the alveoli, but also in the finer bronchi. In one case Friedlaender found the lymph channels of the lung distended with them. They are found in greatest numbers in the period of red hepatization, in lesser numbers in the stages of gray and yellow hepatization. They are usually free, more rarely they are inclosed in cells. The following results were obtained by Sotnischewsky, on chemical examination of a pneumonic lung in the stage of red hepatization:

Water, . . . . .	73.56
Solid substances, . . . . .	21.44
<hr/>	
Organic matters, . . . . .	20.74
Inorganic matters, . . . . .	0.74

In addition to leucin, tyrosin, xanthin, taurin, glycogen, cholesterin, and fatty acids, the watery extract contained an albuminoid substance, which coagulated at 55°, and differed from myosin and globulin substances inasmuch as it was not precipitated on boiling. The watery extract also presented distinct peptone reaction.

The first changes generally appear at the hilus, and the inflammation then spreads to the surface of the lung. Cases in which the inflammation occurs only within the lung, and is surrounded on all sides by aerated lung tissue, are comparatively rare (central pneumonia).

The site of inflammation may be recognized almost always on external inspection of the lung. The lung appears larger, and more or less deep, parallel furrows, produced by the pressure of the ribs, are often seen upon the surface. At the periphery of the inflammation, the lung is usually darker, bluish or blackish red. On palpation, the tissue feels firm and empty of air, and crepitation is entirely absent. The weight of the lung is more or less increased.

I have found the following weights in some of my cases: *a. Man*, æt. 36

years; total left-sided pneumonia; weight of right lung 740 gm., of left lung 2,257 gm. *b.* Man, æt. 50 years; pneumonia of left lower lobe; weight of right lung 460 gm., of left lung 967 gm. *c.* Man, æt. 45 years; total right-sided pneumonia; weight of right lung 1,121 gm., of left lung 640 gm.

It must be remembered that the right lung normally weighs about 82 gm. more than the left.

Gendrin found the specific gravity of the diseased lung changed in the relation of 19 to 1.

The pulmonary pleura over the inflamed district is almost always destitute of gloss and opaque, not infrequently presents subpleural extravasations, and is usually covered with fibrinous membranes. Hence the majority of cases of fibrinous pneumonia are cases of fibrinous pleuro-pneumonia. In rare cases there is an accumulation of serous, sero-fibrinous, or purulent exudation.

The bronchi are usually in a condition of catarrhal inflammation. The finer bronchi, on the other hand, are generally filled with fibrinous coagula. In rare cases the entire bronchial tree is found in a condition of fibrinous inflammation (pneumonia massiva).

The bronchial glands are generally swollen, reddened, and unusually succulent.

As a rule, the bodies of pneumonic patients are not strikingly emaciated. Rigor mortis is well marked. Numerous *livores mortis* on the dependent parts of the body. The muscles are often dry, of the color of ham. Waxy degeneration of the muscles may be found on microscopical examination.

The right heart is distended with blood, the left heart is empty. The blood in the heart consists of loose cruor clots, often also of yellowish fibrinous clots. The heart muscle is sometimes extremely flaccid, brittle, and pale. In the majority of cases the microscope reveals no changes, but sometimes the muscular fibres are granular in places, and individual fibres may present fatty degeneration.

As a rule, the abdominal organs are found in a condition of passive congestion. The liver is enlarged, often gorged with blood, cloudy, and swollen. The microscopical changes are similar to those in the heart. The spleen is often enlarged, soft, and succulent (spleen of infection). The kidneys also present hyperæmic, in some cases parenchymatous changes; there is often catarrh of the mucous membrane of the pelvis of the kidney and of the ureters. The intestinal mucous membrane is generally in a condition of venous congestion and catarrhal swelling, and the follicles are not infrequently enlarged and swollen. The brain presents, as a rule, venous congestion, more rarely anæmia.

III. SYMPTOMS.—Genuine fibrinous pneumonia usually begins suddenly with a vigorous chill. More rarely it is preceded for several days by prodromal symptoms, consisting of general malaise and bodily and mental torpidity. The chill often appears in the midst of work, or the patients are roused from profound sleep by the violent shaking, chattering of the teeth, and feeling of coldness. It lasts generally from half an hour to several hours, and gradually gives way to a feeling of increased heat, which is felt first in the interior of the body, and then spreads over the surface. The rectal temperature is generally elevated during the chill.

The more the sensation of heat increases, the more marked are the other febrile symptoms; reddened face, glistening, often staring eyes, increased thirst, vague pains, and tired feeling in the limbs, acceleration of the pulse, diminished diuresis, and high color of the urine.

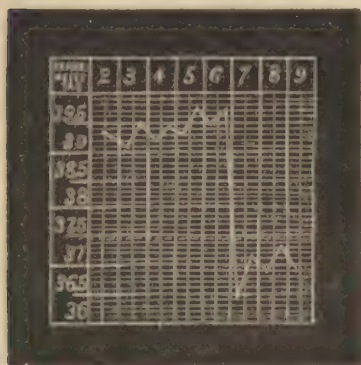


As a general thing, certain subjective complaints, which arouse a suspicion of pulmonary disease, very soon make their appearance. The patients often complain of fluttering in the chest and a sticking pain. Cough and expectoration make their appearance. The sputum, which is at first tinged and streaked with blood, later assumes a more diffuse rusty color. The latter sign renders the diagnosis almost absolutely certain. The respirations are accelerated, irregular, often gasping, and attended with moaning. Speech is interrupted.

The first physical changes in the lungs are not demonstrable, as a general thing, until the end of the first twelve or twenty-four hours. They begin with the evidences of the presence of fluid in the alveoli, then change to those of the absence of air in the pulmonary parenchyma, and close with the return of fluid in the alveolar spaces. But as the changes of various stages of hepatization are generally present at the same time, we must be prepared to find manifold phenomena on physical examination.

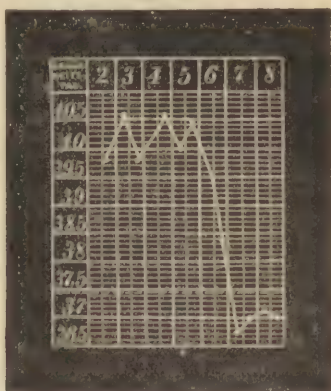
The favorable termination of the disease begins with the sudden change of the general symptoms, the bodily temperature falling in a few hours to the normal or even below the normal. This process, which is associated with other favorable changes, and usually occurs from the fifth to eighth day, is known as the crisis. It is soon followed by a reso-

FIG. 71.



Temperature curve in genuine fibrinous pneumonia in a man set. 43 years.

FIG. 72.



Temperature curve in fibrinous pneumonia with protracted crisis in a man set. 37 years.

lution of the local changes in the lungs, and under favorable circumstances the *restitutio ad integrum* is completed at the end of the second week.

We will now enter more fully into the details of this hasty sketch.

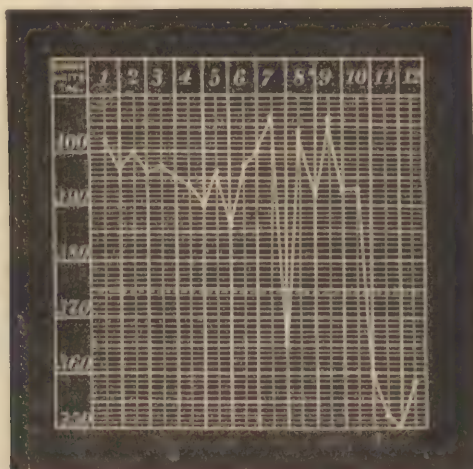
The lower lobe of the right lung is affected most frequently. It has been supposed that this is owing to the fact that the right bronchus is larger, and the power of aspiration of the right lung is greater than that of the left, so that the infecting bodies are drawn more readily into the right lung and then, by force of gravity, into the lower lobe. The remaining lobes are affected in the following order of frequency: left lower lobe, right middle lobe, right upper lobe, and left upper lobe. Bilateral pneumonia is not frequent. The disease often begins in one lobe and gradually spreads over the entire lung (total pneumonia), or it extends to the other lung.

*a. Fever.*—The bodily temperature usually ranges between  $39^{\circ}$ – $41^{\circ}$  C. As a rule, the fever is continued, *i. e.*, the difference between the morning and evening temperature is not more than one degree. Greater variations sometimes occur from the third to the fifth day, and indicate the impending cessation of the fever. As a rule, the fever ends suddenly from the fifth to the eighth day. The temperature curve thereby is made characteristic, that the diagnosis may be made from it alone.

The phenomena attending the sudden fall of temperature are termed the crisis. It has been noticed that the crisis appears somewhat more frequently on the uneven days. If the crisis occurs on the second or third day of the disease, we must expect another rise of temperature in the next few days, and that the permanent fall will not take place until several days later. If the temperature remains high for more than two weeks, a gradual defervescence (lysis) often occurs, or some complication is present.

As a rule, the crisis begins towards evening or during the night, more rarely in the morning. It is often completed in six to twelve hours. In

FIG. 73.



Temperature curve in fibrous pneumonia in a girl *æt.* 5 years. Pseudocrisis on the 7th day, definitive crisis on the 10th day. After Gerhardt.

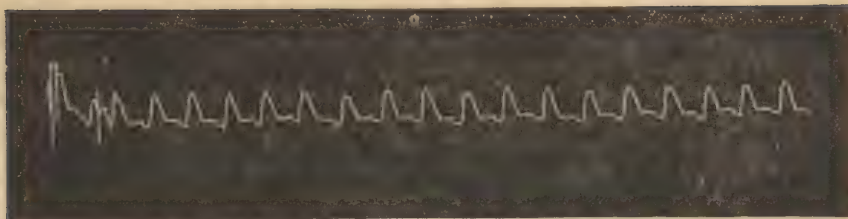
one case Juergensen observed a rectal temperature of only  $35.3^{\circ}$  C. immediately after the crisis.

A period of thirty-six hours sometimes elapses before defervescence is completed (protracted crisis). The fall of temperature may be uninterrupted in such cases (Fig. 72) or a slight exacerbation may be noticed on the evening before permanent defervescence.

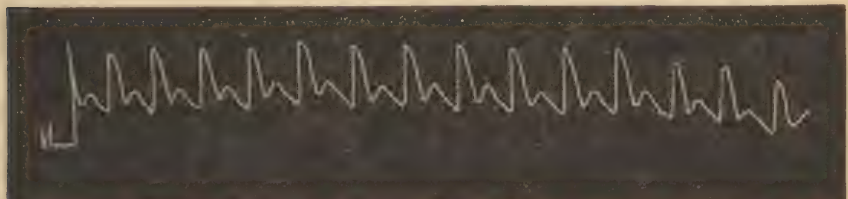
The occurrence of the crisis is sometimes preceded by a brief excessive rise of temperature and an exacerbation of the general symptoms (sudden occurrence of delirium, convulsions, occasionally chilly sensations and chills). These symptoms are devoid of danger.

Pseudo-crisis is a term applied to a febrile curve in which a considerable fall of temperature occurs, but is followed by another rise, the temperature not falling permanently to the norma until after the lapse of a few days (Fig. 73).

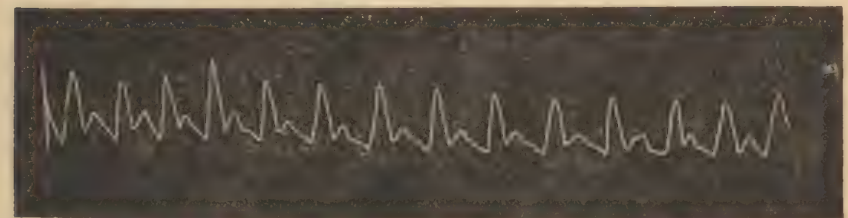
FIG. 74.



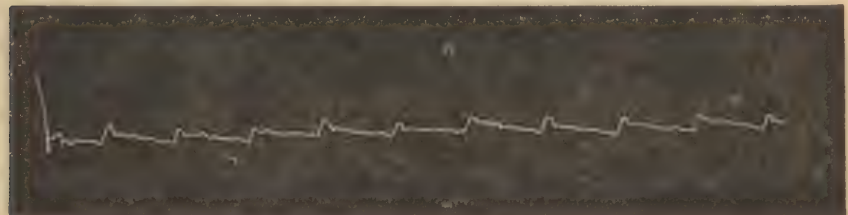
2d day.—Morning: T. 40.0°, P. 112, R. 36. Noon: T. 40.2°, P. 116, R. 40. Night: T. 40°, P. 108, R. 36.



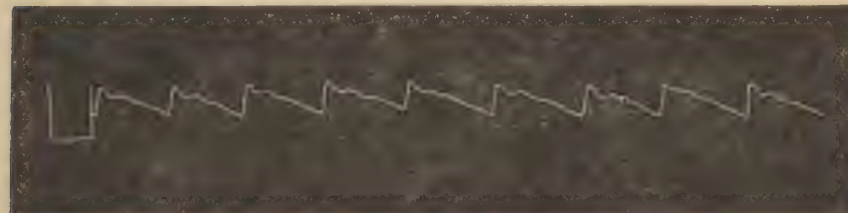
3d day.—Morning: T. 39.4°, P. 92, R. 28. Noon: T. 39°, P. 84, R. 28. Night: T. 38°, P. 84, R. 28.



4th day.—Morning: T. 39°, P. 92, R. 32. Noon: T. 38.6°, P. 96, R. 28. Night: T. 38°, P. 92, R. 28.



5th day.—Morning: T. 36.5°, P. 84, R., 28. Noon: T. 36.3°, P. 88, R. 24. Night: T. 36.6°, P. 68, R., 24.



6th day.—Morning: T. 36.4°, P. 92, R. 28. Night: T. 36.8°, P. 80, R. 20.

Pulse curve of the right radial artery in fibrinous pneumonia in a man æt. 27 years. All the sphygmographic tracings were taken at 9 A.M.



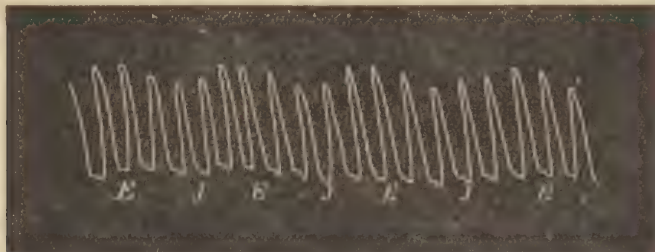
During the fall of temperature profuse perspiration makes its appearance. The hitherto restless and sleepless patient falls into a profound, refreshing sleep, and awakes with a feeling of great relief. At the same time the pulse becomes slower. Not infrequently the urine for the first time deposits a red sediment of uric acid salts. In addition, there are changes in the chemical constitution of the urine (vide page 303).

*b. Pulse.*—The frequency of the pulse increases with the primary rise of temperature. In adults it is generally 100–120 a minute. If it exceeds 130 a minute, the disease is very serious, and when it rises above 140, the prognosis is usually very grave. In children the pulse may rise to 200 beats a minute. The acceleration of the pulse is the result, in part, of the increased bodily temperature, in part of the mechanical disturbance of the pulmonary circulation. With the beginning of the crisis the frequency of the pulse also diminishes and may become subnormal (30–40 a minute). If a slow pulse is found at the height of the fever, special complications (cardiac or cerebral disease) must be suspected.

As a rule, the pulse is regular. Irregularity and intermittence of the pulse should put us on our guard, as this is often the result of serious disturbances of the innervation and energy of the heart. Stricker described *pulsus bigeminus* in one case. Insignificant irregularities of the pulse may appear shortly before and after the crisis.

At the beginning of the disease, the pulse is usually full, large, and hard, *i. e.*, the lumen of the radial artery is large (full), the lateral movements of the arterial trunk are considerable (large), and the wave of blood is with difficulty compressed by the fingers (hard). Towards the end of the disease it grows emptier and smaller. Dirotism is fre-

FIG. 75.



Monocrotic pulse with marked respiratory oscillations in fibrinous pneumonia. T. 40°. E, expiration; J, inspiration. After Bardenheuer and Riegel.

quent and is often most marked immediately before and after the crisis. In one case Cassau found the radial pulse on the affected side smaller than on the healthy side, and he attributes this phenomenon to the pressure of the infiltrated lung on the subclavian artery.

The sphygmographic examination of the radial pulse shows nothing characteristic. The pulse curve is that of fever, the elevation of recoil being very distinct on account of the diminished vascular tension, while the elevation of elasticity disappears more and more (Fig. 74). Respiratory deviations often appear with unusual distinctness (Fig. 75).

*c. Respiration.*—The frequency of the respirations is increased during the period of fever. In adults, 30–40 respirations or more occur during a minute. In children, Juracz has counted 100 respirations a minute.

The respirations are accelerated in part on account of the increased temperature of the body, because the blood then stimulates the respiratory centre and produces so-called heat dyspnoea. The respirations are also accelerated on account of the diminution in the respiratory surface, their superficial character on account of the pains, and occasionally the diminution in the vigor of the heart.

Juergensen attaches great diagnostic and prognostic importance to the disturbance in the relation between the frequency of the pulse and respiration. The normal proportion is 4.5 : 1, but in pneumonia the number of respirations approximates that of the pulse.

*d. Subjective Symptoms.*—Apart from the general febrile disturbance, almost all patients complain of stitches in the chest. These are felt particularly during deep respirations and coughing, so that the patients anxiously attempt to avoid both. The pains are very commonly located in the region of the nipple or at the level of the sixth and seventh ribs, but they may also radiate into the arms. Pain is felt occasionally on the healthy side, sometimes even more severely than on the affected side. These are probably febrile muscular pains.

The cough is generally severe, and, in combination with the pain, not infrequently causes protracted insomnia.

*e. Sensorium.*—Consciousness may be undisturbed during the entire course of the disease. In other cases slight delirium occurs, particularly towards evening when the temperature rises. Or the patients talk and dream a good deal during sleep.

*f. Skin.*—The face is generally very red, the redness often confined almost exclusively to the cheeks. Not infrequently the redness is unilateral, upon the side corresponding to the affected lung.

Cyanosis is not infrequently noticeable upon the lips and nose. At the height of the disease, the skin is dry and hot. Slight diaphoresis occurs occasionally about the third or fourth day. Very profuse diaphoresis occurs at the beginning of the crisis. Cold, clammy sweats are often observed when the termination is unfavorable and collapse is impending.

Special importance is attached to the development of herpes. Herpes labialis is the form usually observed, beginning at the angle of the mouth and spreading upon the vermilion border along a part of the upper and lower lips. It is rarely bilateral, and may then surround the lips and cause them to swell into a shapeless mass. Nasal, auricular, infra-orbital, and supra-orbital herpes is still more rare. Thomas has observed herpes analis, sacro-ischiadicus, and a combination of herpes facialis and herpes manus. The eruption is also observed exceptionally upon the mucous membrane of the mouth.

It develops generally on the second or third day of the disease, in rare cases not until after the crisis. The diagnosis of herpes is easy; clear, yellowish vesicles, about as large as a pin's-head, upon a reddened base, and arranged in groups. At a later period the vesicles become opaque, their contents dry into yellowish or blackish-brown thin crusts, which fall off after a time without leaving a cicatrix. Gerhard attributes them to febrile dilatation of the vessels in the bony canals of the face and irritation of fibres of the trigeminus. But the frequency of herpes in pneumonia, and its absence in typhoid fever, is inexplicable on such a theory. Herpes has a favorable prognostic significance, inasmuch as it usually develops when the pneumonia is uncomplicated. It is also important in the diagnosis of certain cases, for since it is hardly ever ob-

served in typhoid fever, its presence in doubtful cases would decide in favor of pneumonia.

Erythema, roseola, urticaria, pemphigus, acne, and purpura have also been noticed in rare cases. The development of miliaria presents no significance, inasmuch as it usually follows diaphoresis.

*g. Position of the Body.*—The position of the body is not always constant, and depends in part upon the subjective symptoms. If the pains during respiration are very considerable, the patients often lie upon the affected side in order to moderate the respiratory movements on that side. In this way, also, the healthy side is unimpeded in its movements. Together with the decubitus on the diseased side, we often find that the upper part of the spinal column is curved towards the same side, so that the intercostal spaces are narrowed, and the spine forms a convex curve towards the healthy side. As a matter of course, this impedes still more the mobility of the affected side. Dorsal decubitus, however, is observed not infrequently.

*h. Local Thoracic Changes.*—On inspection, the respiratory movements of the affected side almost always appear to be diminished. This side is either motionless or the movements are less vigorous, not infrequently delayed and irregular (effects of the pleuritic pain). The parts which are not directly affected act so much more vigorously (vicariously) or there is an unusual degree of diaphragmatic respiration.

The results of palpation are very important. Vocal fremitus over the pneumonic lung is increased as soon as the alveoli are filled with solid exudation, and the conditions are thus rendered more favorable for the conduction of waves of sound from the interior of the bronchial tree to the wall of the thorax.

In order to avoid mistakes, it should be remembered that the vocal fremitus is normally somewhat more marked on the right side than on the left. The increase of vocal fremitus in pneumonia will be absent if the main bronchus leading to the infiltrated lung is occluded by mucus or other substances. However, its absence is usually temporary in such cases, and the vocal fremitus is again increased after vigorous coughing, which removes the secretion and restores the permeability of the bronchi. Gerhardt found that the increase of vocal fremitus may be absent if the infiltration is very extensive, perhaps because the inner surface of the thorax is put too strongly on the stretch, and thus partly prevents the conduction of the waves of sound. If the pneumonia becomes complicated with fluid pleurisy, the vocal fremitus gradually diminishes, and may become less than normal if the amount of fluid is sufficient.

Measurement shows an increase of 0.5 to 2.5 cm. in the circumference of the affected side of the chest.

With the aid of the pneumatometer, I found that the vigor of inspiration and expiration, especially of the former, is diminished during the existence of pneumonia.

A local elevation of temperature is observed sometimes, though not constantly, in the axilla of the affected side or upon the chest-wall.

The phenomena observed on percussion vary according to the different stages of the disease.

During the stages of engorgement and resolution, the percussion sound is tympanitic, on account of the relaxation of the pulmonary parenchyma, which is filled with a fluid containing air. Cracked-pot resonance is heard occasionally, but its origin has not been explained satisfactorily. If the exudation in the alveoli has become non-aërated and firm (stage of hepatization) the percussion sound is dull. As a matter of course, the non-aërated district must lie sufficiently near the surface, and pos-



sess certain dimensions, before the percussion sound becomes dull. If the infiltration is more than 5 cm. below the surface of the lung, it can hardly be recognized by percussion, and in general, vigorous percussion is necessary in order to recognize spots of inflammation which are surrounded by pulmonary tissue containing air. Peripheral foci must have at least a circumference of 5 cm. and a thickness of 2 cm. in order to be recognizable by percussion, and, as a matter of course, require gentle percussion. In all cases, it is well to employ Wintrich's palpatory percussion, and to pay attention to the feeling of increased resistance over the diseased lung.

During the stage of hepatization, Baeumler occasionally noticed a very resonant tympanic sound when a thin layer of aerated parenchyma was situated over a non-aerated district. During the stage of hepatization, we occasionally hear a tympanic sound, which becomes higher on opening the mouth, lower on closing the mouth. In these cases, there is generally an extensive pneumonia of the upper lobe, so that the vibrations produced by percussion are propagated to the column of air in a main bronchus, and produce waves of sound in it. Jürgensen observed this phenomenon occasionally in pneumonia of the lower lobes. Stern found a metallic percussion sound in four cases. Very extensive disease was present in these cases, all of which proved fatal, but Skoda has seen recovery under similar circumstances.

The percutory phenomena present gradual transitions, not alone in time, but but also in place.

During the stages of engorgement and resolution, fine crepitant râles indicate the presence of fluid in the alveolar spaces. In hepatized districts bronchial breathing is heard, because the non-aerated alveoli have lost the power of converting the bronchial breathing, which is conveyed from the larynx, into vesicular breathing. At the same time the râles developing in the bronchi which are filled with fluid (so far as the bronchi are surrounded by parenchyma destitute of air) assume a consonant character. The bronchophony is increased, and ægophony is heard not infrequently. Bacelli's phenomenon (audible whispered voice) is observed not very rarely.

Crepitant râles are almost always heard only during inspiration, often only on deep inspiration and towards the close of the act. They sometimes disappear after repeated deep respirations and only reappear after a time. They are produced by the tearing of the alveolar walls from their fluid contents. Penzoldt has recently described some cases of inspiratory and expiratory or exclusively expiratory crepitant râles.

*i. Sputum.*—The character of the sputum possesses great diagnostic importance, since a rusty sputum is hardly ever observed except in fibrinous pneumonia. In the beginning of the disease, the sputum is usually tough, mucous, colorless, and contains a few specks and streaks of blood. In a very short time it assumes the color of fresh iron rust. It is extremely tough, so that some patients cannot expectorate it, but must remove it from the mouth with the aid of the fingers. It is slightly frothy, vitreous, and transparent despite the rust color. It varies generally in amount from 30–200 ccm.

Fibrinous bronchial casts, which originate in the finest ends of the bronchi, are found almost constantly in the sputum. They sink to the bottom, where they form grayish white lumps or rolled up threads. If the latter are shaken in water, they are resolved into cylindrical and dichotomously branched structures, corresponding to the ramifications of the finer bronchi (Fig. 76). At the sites of bifurcation they are not infrequently widened, sometimes they contain swellings

which are produced by air bubbles. Nodular swellings, corresponding to the alveoli, are found occasionally at the finer ends. The bronchial casts belong to the stage of hepatization, appear generally on the third day of the disease and disappear on the seventh day. In one case Remak observed them as late as the fourteenth day, and Biermer found them even in the third week. The latter writer estimates that as many as thirty are found in a single day.

The small fibrinous bronchial casts are almost pathognomonic of pneumonia. Laennec observed them in a case of phthisis, and they also occur in primary fibrinous bronchitis.

Vierordt and Jacksch found spirals in the sputum, during the stage

FIG. 76.



Fibrinous bronchial casts from the sputum of fibrinous pneumonia. Natural size.

of resolution, similar to those present in asthma. According to my own experience, the sputum of fibrinous pneumonia contains spirals not infrequently.

On microscopical examination it is found to contain round cells, alveolar epithelium, intact or swollen red blood-globules, ciliated epithelium from the bronchial mucous membrane (Fig. 77). Ziehl first demonstrated the presence of pneumococci, and we can confirm their almost constant occurrence in the rusty sputum. Sometimes they may be absent in several preparations, in others they are found in large masses. Similar cocci, however, are found in the sputum of other diseases, and this is readily understood in view of their presence in the buccal cavity. In order to make a preparation of the pneumococci, a bit of sputum, half as large as a pin's head, is removed by means

FIG. 77.



Rusty sputum of fibrinous pneumonia in a man æt. 37 years. Third day of the disease. The majority of the red blood-globules are lentil shaped. Enlarged 275 times.

of a needle which has been made red hot and carefully cleaned, and then

FIG. 78.



Pneumococci from the rusty sputum of fibrinous pneumonia. Third day of the disease. Methyl blue preparation. Enlarged 600 times. Water immersion.



compressed between two cover glasses into a uniformly thin layer. The cover glasses are then separated from one another, grasped with a pair of forceps and drawn five to ten times through the flame of a spirit lamp, the side to which the sputum is adherent being directed upwards. The surface to which the sputum is adherent is now allowed to float for twenty-four hours upon a concentrated solution of a basic anilin color—fuchsin, gentian violet, methyl blue, dahlia. The cover glass is then removed and carefully washed in absolute alcohol. It is then rapidly washed in distilled water, dried by being drawn through the flame of the spirit lamp, and placed in Canada balsam. The pneumococci are then recognizable by their rounded or oval shape and the clearer, slightly stained gelatinous capsule. Single cocci are found rarely, more frequently two are situated in a common capsule (diplococci); even three, four, or more may be found together.

The fibrinous bronchial casts consist of a fibrinous mass arranged, in great part, in parallel fibres and containing, in places, fatty granules and white and red blood-globules. Ciliated epithelial cells are found occasionally upon the outer surface of the casts. In addition, the sputum sometimes contains casts which seem to possess more of a membranous character and to be imperfectly developed. I suspect that they are closely related to the previously mentioned spirals.

The rusty color of the sputum is the result of chemical changes in the coloring matter of the red blood-globules. Juergensen claims that he has seen the rusty sputum in miliary tuberculosis of the lungs, but this does not impair its diagnostic importance in fibrinous pneumonia. It should not be mistaken for the brown color of the sputum in hemorrhagic infarction.

Renk gives the following results of the chemical analysis of the sputum in two cases of pneumonia:

	CASE I.	CASE II.
Water, . . . . .	90.99	96.36
Solid substances, . . . . .	9.01	3.64
<hr/>		<hr/>
Solid substances, . . . . .	8.35	2.76
Inorganic substances, . . . . .	0.66	0.88
<hr/>		<hr/>
Mucin, . . . . .	1.28	1.09
Albumin, . . . . .	3.09	
Fat, . . . . .	0.032	0.02
Extractive matters, . . . . .	3.95	1.65

As the pneumonia approaches resolution, the color of the sputum becomes citron or saffron yellow. It also becomes more profuse, and finally assumes the appearances of ordinary muco-purulent expectoration.

The red blood-globules of the yellow sputum are less in number, paler, more swollen, and partly disintegrated. The mucous corpuscles and alveolar epithelium are in a condition of fatty degeneration. Granular and fatty detritus is present in abundance.

*k. Heart.*—During the fever, cardiac dulness is often increased on the right side (febrile dilatation of the thin right ventricle, associated with increased pressure in the pulmonary artery). The diastolic pulmonary sound is often intensified.

*l. Spleen and liver* are not infrequently enlarged.

*m. The tongue* is usually coated grayish-white or yellow, and is dry, fissured, occasionally bloody, or covered with a fuliginous blackish-red

coating. Anorexia and increased thirst are present. There is usually a tendency to constipation.

*n. Urine.*—The urine is scanty, reddish, very acid, has an increased specific gravity, and not infrequently contains traces of albumin, and a few, usually hyaline, casts. A deposit of urates is observed when the urine is concentrated.

The urea is generally increased threefold, but there is no relation between the height of the fever and the amount of urea. Shortly before the crisis, the amount of urea diminishes; after the crisis, the amount becomes excessive. Fraenkel assumes that the excretory functions of the kidneys are disturbed during the fever, an accumulation of urea in the body occurs, and is not voided until the fever subsides. On the other hand, Schultzen proposes the theory that only a portion of the albumin, which is received into the circulation during the fever, undergoes retrogressive metamorphosis, another portion being retained in the blood until after defervescence.

The uric acid, ammonia, and kreatinin also increase in quantity, but the chloride of sodium diminishes, until only a trace is left at the height of the fever. The relative quantity of phosphoric acid is also diminished, with the exception, according to Raineri Boffito, of magnesia phosphate. The absolute amount of sulphuric acid excreted is increased.

The description just given refers only to uncomplicated cases. But variations are often observed, sometimes as the result of an unusual course of the disease, sometimes from individual peculiarities, sometimes from a different degree of infection, and finally, from complications proper.

With regard to the course of the disease, we distinguish, apart from the typical cases, ephemeral, protracted, spreading, wandering, relapsing, intermittent, and apyrexial fibrinous pneumonia.

*a.* In ephemeral pneumonia, the febrile process does not last more than a day, and the local symptoms also subside in a few days. Wunderlich, Leube, and Weil have described cases of this kind.

*b.* Abortive pneumonia is that form in which, despite well developed general symptoms, the local changes are not completely developed. Engorgement and rusty sputum are present, but the signs of hepatization remain absent.

*c.* In protracted pneumonia, the local and general symptoms persist unusually long. The fever extends into the second, even into the third week, the temperature then falls gradually, and the local changes may be demonstrable for four to six weeks, or even longer.

*d.* Spreading pneumonia is characterized by the propagation of the inflammation by continuity, so that an originally circumscribed affection gradually invades the greater part of one lung.

*e.* In wandering pneumonia (*pneumonia migrans*), portions of the lung which are separated from one another are affected by the inflammation. Frequently, only the stage of engorgement occurs, but the development of each new focus of disease is shown by an exacerbation of temperature. Furthermore, a diseased district may again undergo inflammation. The disease may be protracted over a very long period (three months in a case reported by Waldenburg). Bruzelius states that he has observed wandering pneumonia a number of times in acute articular rheumatism, and Vaillard makes a similar statement. Waldenburg also calls attention to the similarity of its course with erysipelas migrans,

while Friedreich emphasizes the fact that pneumonia migrans is more frequent during an epidemic of erysipelas. It has, therefore, been supposed that it forms a sort of internal erysipelas. It is said to be distinguished from non-erysipelatous pneumonia by enlargement of the spleen.

*f.* In relapsing pneumonia, chill, fever, and local signs of inflammation suddenly reappear a few days after the febrile symptoms have ceased and the local changes have resolved. The second inflammation may develop at the site of the former one, or in a hitherto unaffected spot.

*g.* Intermittent pneumonia develops under the influence of the malarial poison, almost exclusively, therefore, in marshy and malarial regions. Frison states that it is most frequent in spring and autumn and during a period of great changes in temperature. The patients have generally had several attacks of quotidian or tertian intermittent. Then there is a sudden appearance of chill, fever, pain in the chest, dyspnoea, cough, and crepitant râles, which disappear at the end of a few hours. Such attacks recur at certain hours of the day, and after several attacks there are permanent symptoms of infiltration. The left lower lobe is said to be affected with special frequency, but Weinlechner described a case in which a different part of the lung was affected at each attack. The disease may be relieved by the use of quinine, otherwise death is almost inevitable. The fatal termination occurs with symptoms of suffocation, especially when the disease is complicated with pleurisy or pericarditis, or severe cerebral symptoms close the scene.

*h.* Apyrexial pneumonia is extremely rare. Cases have been reported by Wunderlich, Kerányi, and Mazzetti. A few weeks ago, pneumonia developed in one day in three patients in my hospital wards, and all ran an apyrexial course. In addition to the local changes in the lungs, the patients presented the rusty sputum, which contained pneumococci. The patients had been in the hospital for months, were in separate wards, and there was no pneumonia in the hospital at the time.

The individual conditions are especially important in the fibrinous pneumonia of children, old people, and drinkers, and in secondary fibrinous pneumonia.

*a.* In the pneumonia of childhood, the initial chill is absent in the majority of cases. In its stead we notice great apathy and drowsiness, together with a pale and cool skin, or violent and repeated vomiting, or an epileptiform seizure. During the period of fever, there is a great tendency to cerebral symptoms: delirium, general convulsions, twitchings in the limbs. A marked crisis is often absent and lysis occurs more frequently than in adults. Relapses also appear to occur more frequently in children. Children hardly ever expectorate; they swallow the sputum.

*b.* The pneumonia of old age not infrequently develops insidiously: and since the fever and subjective symptoms may be very slight, it may easily happen that old people die in collapse from an unknown cause which, upon autopsy, presents itself as a terminal pneumonia. It has been held that fever is often absent in the pneumonia of old age, but this is only true if we rely solely upon the axillary temperature. Bergeron showed that there is not infrequently a difference of 2-3° C. between the axillary and rectal temperatures in old people.

The pneumonia of old people also presents an adynamic or asthenic character. The patients rapidly decline, present evidences of heart failure, and often die with surprising rapidity, and occasionally quite suddenly. Expectoration may be absent in them as it is in children. The anatomical changes not infrequently develop with extraordinary slowness, and the hepatization is characterized by unusual flaccidity.

*c.* Pneumonia of drunkards is often observed, since the organism, when depraved by the abuse of alcohol, exhibits a marked predisposition to this disease. It is often situated in the upper lobes, and resolves very slowly. An outbreak of



delirium tremens threatens during the period of fever. A careful pulmonary examination should be made of all delirious patients who suffer from fever, since in many of these cases the pneumonia may be unattended by cough, dyspnoea, or other suspicious subjective symptoms. The pneumonia of drunkards, like that of old age, is especially dangerous, on account of its asthenic character (mainly the result of fatty heart) and sudden fatal collapse is not very infrequent.

*d.* In secondary pneumonia, the slow development should be specially considered. In this form, also, there is danger of asthenic symptoms, because the organism is enfeebled by previous disease.

Independently of individual conditions, genuine fibrinous pneumonia may run an extremely dangerous course, on account of the specially severe form of infection. Here rapid loss of power, and particularly the weakness of the heart muscle, are the main features, and the evidences of epidemic spread and infection have been demonstrated more frequently and distinctly than usual in this very form of pneumonia (primary asthenic pneumonia, typhoid, putrid, malignant, or bilious pneumonia).

The disease often begins with prodromata of several days' duration. The initial chill may be absent. The fever is usually very marked, and excitement and delirium, or an apathetic typhoid condition are prominent features. The general appearance shows that the patient is very seriously ill, the vital energies are rapidly exhausted. The local changes often do not develop for a long time, and are frequently situated in the upper lobes or in both lungs. Sputum may be entirely absent, or it is markedly hemorrhagic. Enlargement of the spleen and liver can usually be demonstrated. Albuminuria, sometimes very considerable, is almost always present. The conjunctiva and skin are often jaundiced. The disease is usually epidemic and its spread is favored by bad hygienic conditions. It terminates often in pulmonary abscess or gangrene, is often associated with complications (pericarditis, meningitis, mediastinitis, gastro-enteritis, etc.), and often proves fatal. It is observed more frequently during the summer and autumn than in the spring.

Fibrinous pneumonia is sometimes associated with complications.

The most frequent ones are bronchitis and pleurisy. Indeed, they can hardly be called complications, at least this is true of fibrinous bronchitis of the finer bronchi and of dry pleurisy. Bronchitis of the large bronchi and fluid pleurisy constitute real complications which protract the course of the disease, and make it more serious.

Pericarditis is associated most frequently with left-sided or bilateral pneumonia. Inflammation of the lingual process of the left lung has a special significance in this respect. It should not be mistaken for pleurisy, and we must be careful not to interpret an infiltration of the median borders of the lung as enlargement of cardiac dulness by pericarditic exudation.

Endocarditis occurs much more infrequently. As a matter of course, not every systolic murmur may be attributed to endocarditis, because it may be of febrile origin. The evidences of cardiac dilatation and hypertrophy must be present in order to make the diagnosis positive.

Kühn described a complication with mediastinitis in a case of infectious pneumonia.

Jaundice occurs with extraordinary frequency during the course of fibrinous pneumonia, but in some cases it is unimportant, in others it possesses a very serious import. In certain cases it is the result of disturbances in the respiratory movements. As the discharge of bile is

favoured by the movements of the diaphragm, it is evident that, when these are impeded, stasis of bile may result.

This is particularly true of right-sided pneumonia, especially when diaphragmatic pleurisy, which sometimes gives rise to perihepatitis, is also present. Jaundice is occasionally the result of circulatory disturbances, inasmuch as the infiltrated lung impedes the flow of blood from the inferior vena cava and the hepatic vein. Sometimes the jaundice is simply the result of gastro-duodenal catarrh. Under such circumstances, the stools may lose their color. At no times is jaundice a desirable complication, since, in addition to biliary pigment, the biliary acids are also taken up by the blood. These constitute a dangerous heart poison—a fact which is especially dangerous in pneumonia, in which *per se* insufficiency of the heart is to be dreaded. Finally, in another group of cases, the jaundice is the result of dissolution of the blood. In all probability this is a form of hæmatogenous icterus, which results from direct transformation, within the vessels, of the pigment of the dissolved red blood-globules into biliary pigment. This form is apt to complicate asthenic pneumonia. If a sufficient amount of biliary pigment passes into the sputum, the latter assumes a grass-green color, and the well-known Gmelin reaction follows the addition of impure nitric acid.

Gastric and intestinal catarrh also forms a serious complication. It not alone accelerates the exhaustion of the vital energies, but experience also teaches that it is apt to complicate asthenic pneumonia. In certain cases the catarrh appears only at the beginning of the disease and takes the place of the chill. This is very rare in adults, much more common in children.

Slight albuminuria is a symptom of fever and possesses no significance. Warfvinge observed it in forty-seven per cent of his cases. Marked albuminuria and the presence of numerous casts in the urine are indicative of more serious disease of the kidneys, usually of acute diffuse nephritis. It is a striking fact that the symptoms of nephritis often disappear with great rapidity, whether the fever and local pulmonary changes undergo exacerbation or improvement. Urine which contains albumin, casts, and a large number of blood-globules to-day, is perhaps entirely free from them to-morrow. The changes usually disappear in five to seven days. (Edema is produced only in exceptional cases. It has not been positively determined that the disease ever passes into the chronic form of Bright's disease. Hæmaturia is observed occasionally. On the whole, severe renal complications are observed usually in severe or asthenic forms of operations.

Disturbances of the nervous system are frequent, and depend partly on individual conditions (children, old people, drinkers), partly on the height of the fever or the severity of the infection. In drunkards we should always be mindful of the danger of the development of delirium tremens. Fibrinous pneumonia also bears intimate relations to purulent meningitis. The meningitic symptoms rarely develop at the beginning of the pneumonia, but usually from the third to the eighth day (in one case during the fourth week). Immermann, Heller, and Maurer described cases which occurred while cerebro-spinal meningitis was epidemic. Meningitis has also been observed a number of times as a complication of asthenic pneumonia. Willich describes a case in which the meningitic symptoms preceded the pneumonia. Nauwerck has recently reported a series of cases and attempts to prove that the cerebral affection is the result of embolic processes following purulent

infiltration of the lungs, thrombosis of the pulmonary veins, disintegration of vegetations in the heart, or ulcerative endocarditis. It is much more probable that the meningitis is caused by pneumococci, and Eberth has found schizomycetes in the purulent exudation on the meninges.

The symptoms should be carefully considered, since the meningitic phenomena are not always clearly defined. Death sometimes occurs in deep coma or wild delirium. On the other hand, we sometimes notice cerebral symptoms which are not the result of palpable lesions of the meninges. A short time ago I treated a boy, æt. seventeen years, who was suddenly seized with a chill at night; on the following morning considerable fever and profound coma; death in thirty-six hours. At the autopsy, congestion of the meninges and beginning hepatization of the lower lobe of the right lung.

Steiner states that meningitic symptoms sometimes appear in children if otitis interna has developed at the same time as the pneumonia.

Hemiplegia develops occasionally during the course of pneumonia. This takes place generally in old people, and is probably the result of atheroma and thrombosis of the cerebral arteries, with subsequent softening of the brain. Such cases almost always terminate fatally. The paralysis is occasionally temporary and probably of vaso-motor origin. Kussmaul observed the development of hemiplegia in the night, after a subcutaneous injection of morphine had been made the same evening; the patient recovered entirely. Epileptiform attacks have been observed at times.

Disturbances of vision (amblyopia, chromatopsia) have also been noticed. Roque described dilatation of the pupil on the affected side.

Lesions of the skin are among the rarer complications. Erysipelas, multiple abscesses, gangrene, and noma occur in exceptional cases, as they do in other infectious diseases. Lépine observed unilateral redness and increased heat of the limbs, particularly in old people, and he attributes these symptoms to vaso-motor and reflex disturbances.

I have observed profuse epistaxis a number of times at the beginning of the disease and also at the crisis. Parotitis develops occasionally. Septicæmia and pyæmia may develop during the period of purulent inflammation from absorption of the purulent masses.

The following table (embracing 220 cases, 197 of which occurred in soldiers), collated by Chvostek, shows the frequency of the individual complications:

Meningitis.....	4 times (3 per cent).
Thrombosis of the sinuses .....	1 time (0.5 " " ).
Pericarditis.....	11 times (5.0 " " ).
Endocarditis.....	2 " (1.0 " " ).

The following complications were observed among the soldiers:

Delirium.....	44 times (22.3 per cent).
Pleurisy with effusion.....	31 " (15.7 " " ).
Icterus .....	42 " (21.3 " " ).
Epistaxis .....	6 " ( 3.0 " " ).
Hæmaturia.....	3 " ( 1.6 " " ).

The most favorable and frequent termination of pneumonia is in complete recovery. When death occurs, it is the result of increasing exhaustion, heart failure, œdema of the lungs, or the complications mentioned above. When œdema of the lungs develops, the cyanosis of the skin increases, the skin grows cool and is usually covered with clammy perspiration. Pulse small, pupils contracted; numerous crepitant râles are heard, and the sputum becomes profuse, frothy, thin, and often of



a deep blackish-red color (prune juice expectoration). These conditions often develop with astonishing rapidity.

Among sequelæ of pneumonia are pulmonary abscess, gangrene, retraction, and phthisis.

Paralyses are sometimes left over, as in other infectious diseases. In one case, Leyden observed the symptoms of infantile spinal paralysis; Sinkler reported a case of reflex paralysis (!). Insanity is an occasional sequel.

IV. DIAGNOSIS.—The character of the sputum plays a very important part in the diagnosis of fibrinous pneumonia. If we remember that not every brownish-red or reddish-brown sputum may be termed rust-colored, and if the eye is sufficiently acquainted with the specific color of rust, we may claim that the rusty sputum hardly ever occurs except in fibrinous pneumonia. Its diagnostic significance is especially great in central pneumonia, in which it is the sole local symptom.

As a matter of course, pneumonia may be present even if the sputum is not rusty. When associated with intense jaundice, the sputum has a grass-green color, and this also possesses a valuable diagnostic significance.

Nothnagel and Traube have shown, however, that the green sputum may occur in fibrinous pneumonia uncomplicated with jaundice, if the disease ends in a lysis, not in a crisis, or if it passes into caseation. Traube also observed it temporarily during the termination of pneumonia in pulmonary abscess. Elliot and Janssen found it in sarcoma of the lungs. O. Rosenbach describes a green sputum (?) caused by pigment bacteria.

The examination of the sputum is also important in another respect. The appearance of a saffron-colored sputum indicates beginning resolution, while the prune-juice expectoration should draw our attention to impending œdema of the lungs.

The saffron-colored sputum should be distinguished from the yellow variety, whose yellow color is produced by pigment bacteria, and is found mainly in the superficial frothy layer. It is observed particularly in hospitals in summer, when infection and pigmentation are often conveyed from one spit-box to another (Loewer and Traube).

If expectoration is wanting (in children, old people, drinkers, exhausted individuals), the diagnosis may be impossible in central pneumonia, or may only be made from the fever curve after the cessation of the fever. Even in peripheral pneumonia, none of the physical signs is as characteristic of the disease as the rust-colored sputum. Crepitant râles are also present in œdema and hemorrhagic infarction. Dulness, bronchial breathing, consonant râles, increased vocal fremitus, and bronchophony are also found when the alveoli are filled with cheesy or other non-aërated masses (tumors). Under such circumstances, the diagnosis can only be made from the mode of onset and the course of the disease.

We will again emphasize the fact that we should never be satisfied with an examination of the anterior and posterior surfaces of the thorax, and that not infrequently pneumonia of the right upper or middle lobe begins in the lateral region and even remains localized there.

Pneumonia may be mistaken occasionally for pleurisy with effusion, since dulness, bronchial breathing, consonant râles, ægophony, and Bacelli's phenomenon may also be observed in the latter affection. But the acute onset and acute typical course are absent in pleurisy; the vocal fremitus is diminished in pleurisy, increased in pneumonia; the upper

boundary of dulness is straight or slightly wavy in pleurisy, and in dorsal decubitus is higher near the spine than it is anteriorly; furthermore, in pleurisy the dulness increases from above downwards; it is rarely confined to the upper parts of the thorax, leaving the lower parts free; displacement of adjacent organs, and disappearance of the semi-lunar space on the left side favor the diagnosis of pleurisy; the rusty sputum is absent in simple pleurisy. In very doubtful cases, an exploratory puncture may be made. This is especially important in the differential diagnosis of fluid pleurisy and pneumonia massiva. In the latter disease, the larger bronchi are filled with fibrinous casts, so that vocal fremitus and bronchophony are diminished as in pleurisy. The diagnosis becomes positive even without puncture if large bronchial casts are expectorated, and the vocal fremitus and bronchophony are then intensified.

Pneumonia may sometimes be mistaken for meningitis or typhoid fever, if cerebral or intestinal symptoms and an apathetic condition are prominent. Careful examination of the respiratory organs is necessary in such cases. The presence of herpes would tend to exclude, that of roseola would favor the diagnosis of typhoid fever.

V. PROGNOSIS.—The prognosis is favorable if the patient is young, vigorous, and not addicted to alcohol. It is very serious in children, old people, and drinkers, because the organism is less resistant, and there is danger of insufficiency of the heart's action. For similar reasons the prognosis of secondary pneumonia is more unfavorable than that of the primary form.

But the prognosis depends mainly on the severity of the infection. Primary asthenic pneumonia often presents a terribly high mortality.

The bodily temperature, if it does not go beyond  $40.5^{\circ}$  C., does not affect the prognosis.

The localization of the process sometimes exerts an influence. Unilateral pneumonia runs a more favorable course than the bilateral form. Pneumonia of the upper lobes has an evil reputation, for it is apt to be associated with great febrile disturbance, asthenic symptoms and other severe complications, and often terminates in abscess, gangrene, or caseation.

The prognosis is also determined by the complications. The mortality of 546 cases collected by Warfvinge was 6.2 per cent, that of the cases complicated with delirium tremens was 39.6 per cent, of those complicated with acute enteritis 31.18 per cent, of those complicated with albuminuria 20 per cent.

Finally, the prognosis is grave if the pneumonia has been preceded by other diseases of the lungs (emphysema, phthisis) or heart. Under such circumstances death often occurs from suffocation or paralysis of the heart.

Pregnancy forms a sort of individual complication. Pneumonia is not infrequent in pregnant women and is a source of great danger to the mother and child. Among 26 cases collected by Chatelain, abortion occurred 10 times, premature delivery 9 times. Ten mothers died. The farther advanced the pregnancy, the more probable are premature delivery and death.

The mortality of fibrinous pneumonia varies according to the character of the cases of each individual observer. Fraentzel states that he did not observe a single death among 100 cases occurring in soldiers. For the years 1845–1875 Winger noted in the Christiania Hospital, an

average mortality of 16.8 per cent. In the St. Petersburg Hospital the mortality was 21 per cent.

VI. TREATMENT.—Primary pneumonia runs such a favorable course in vigorous adults, who are not run down by the abuse of alcohol, that special treatment is unnecessary. The patient should be kept in a quiet, roomy apartment, and this should be ventilated morning, noon, and night through an adjoining room. The temperature is kept uniformly at about 15° R., and the air kept moist by means of vessels of water or an inhalation apparatus. The diet should be exclusively fluid (milk, meat soups, eggs, wine) and the thirst may be quenched by the administration of lemonade or acids (muriatic, sulphuric, nitric, phosphoric acid 5.0 : 200., one tablespoonful every two hours, etc.).

When asthenic symptoms appear or in secondary pneumonia, the treatment is entirely different. We should not direct our attention to the local changes, but should remember that pneumonia is a general infection, in which the greatest dangers are threatened on the part of the heart. The main indication, therefore, is to sustain the power of the heart's action.

Above all we must employ large doses of alcohol, and this is done in the pneumonia of drinkers for the further reason that in this way alone can the outbreak of delirium tremens be prevented.

Among the wines to be recommended are those which are specially rich in alcohol, such as sherry, port, marsala, madeira, and certain Rhine wines (Rauhenthaler, Markobrunner, etc.). One-half to one wineglassful may be given every hour. Champagne is also indicated, but the carbonic acid gas should be allowed to escape before being administered, in order to prevent distention of the stomach. The addition of pieces of ice to champagne or white wine increases its stimulating properties.

Too little use is made of alcohol in a more concentrated form. It may be given as brandy (by the tablespoonful every hour), or in the form of a mixture (℞ spirit. dilut., 20.0; Aq. fontan., 180.0; Elix. aurant. comp., 5.0; Syr. simp., 15.0. M. D. S. One tablespoonful every one to two hours. ℞ Spirit. vini dilut., 20.0; Aq. fontan., 180.0; tinct. aromat., 5.0; Syr. simp., 15.0. M. D. S. One tablespoonful every one to two hours).

The form of administration of the alcohol should be changed frequently, in order to prevent the patient from becoming disgusted with it.

It is also important, in asthenic conditions, to reduce the temperature of the body, if it rises above 39° C. According to my experience, no remedy equals antipyrin in the certainty of its effect. 4.0 dissolved in 100. lukewarm water should be injected into the rectum. If no effect is produced in two hours, another enema containing 2.0 should be administered. The administration of 2.0 antipyrin in wafers every two hours until defervescence occurs is less effective because it is apt to produce vomiting, even if 0.01 opium has been added to each dose.

Next to antipyrin in the reliability of its antifebrile action stands kairin. 0.5 is given every hour until the temperature falls below 38° C. and is readministered as soon as the temperature rises above 38° C. But in addition to the inconveniences attending the hourly measurement of the temperature, kairin is not suited to the treatment of pneumonia because it often produces collapse despite every precaution.

Quinine is not a certain remedy, even if 0.5 is given every fifteen minutes until 2.0 to 4.0 have been administered. Vomiting is often noticed after the first doses, but this ceases, as a rule, if its administration is



continued. Gerhardt recommends inhalations of quinine, employing one-third the quantity which is usually given internally. It may also be employed subcutaneously (chinin. muriat., glycerin. pur., aq. destil.,  $\text{ãã}$  5.0. M. D. S. One syringe-ful subcutaneously: the mixture, in which the quinine is deposited as a white crystalline precipitate, must first be placed in hot water until the crystals have entirely dissolved). Quinine may also be administered by enema (2.0 with three tablespoonfuls starch flour and five tablespoonfuls of lukewarm water, slowly injected into the rectum).

Salicylic acid and salicylate of sodium, in large doses (5.0–10.0), will often produce a marked fall of temperature and early appearance of the crisis, but they should be given with great caution, on account of their tendency to produce collapse. In the Berlin City Hospital, about twenty-six per cent of the cases died under the salicylic acid treatment.

Benzoate of sodium (5.0–10.0 at a dose) is less apt to produce collapse, but is also much less certain in its effects.

Tartar emetic, veratrine, and digitalis act as antipyretics only by diminishing the vigor of the heart's action and producing collapse, and should not be employed in pneumonia. This is also true of venesection.

The antipyrexial action of antipyrin is so certain that it is generally unnecessary to employ cold and cool baths. We employ baths in fibrinous pneumonia, not to combat the fever, but to obtain a stimulant effect. If necessary, a bath at 28° R., and lasting ten minutes, should be given morning and evening.

If the asthenic symptoms continue, despite the use of alcohol and the lowering of the temperature of the body, we may prescribe stimulants, for example:  $\text{R}$  Acid. benzoic., 0.3; camphor. tritæ, 0.05; sacch. alb., 0.5. M. f. p., d. t. d. No. x. S. 1 powder every 1–2 hours.  $\text{R}$  Liq. ammon. anisat., 10.0. D. S. 5–10 drops on sugar every hour. If the patient cannot swallow, we may order:  $\text{R}$  Camphor. tritæ, 1.0; ol. amygdal., 10.0. M. D. S. 1 syringe-ful subcutaneously t. i. d.  $\text{R}$  Æther, 10.0. D. S. 1 syringe-ful subcutaneously (this is painful, and not infrequently gives rise to subcutaneous abscesses).

It is sometimes necessary to combat annoying individual symptoms. Violent cough may be relieved by mild narcotics, for example: Aq. amygdal. amar., 10.0; morphin. muriat., 0.1. M. D. S. Ten drops on coughing. If numerous râles and signs of extensive bronchitis are present, we should employ expectorants (vide page 219). The patients are annoyed not infrequently by violent pains in the side, which may be treated with warm compresses, the ice-bag, ten to twenty dry or wet cups, blisters, five to ten leeches, or subcutaneous injections of morphine (morphin. hydrochloric., 1.0; glycerin. puri, aq. destil.,  $\text{ãã}$  15.0. M. D. S. Two to four minims subcutaneously). In insomnia, narcotics are given in the usual doses (morphin. muriat., 0.015, at night.—Chloral hydrat., 3.0; mucilag. salep., syr. rubi idæi,  $\text{ãã}$  25.0. M. D. S. To be taken at night.—Paraldehyde in the same doses), but we would caution strenuously against giving large doses of narcotics, as they are apt to produce symptoms of collapse and suffocation.

The following other methods of treatment may be mentioned: *a.* Schwarz states that the disease may be aborted by the administration of iodide of potassium, if it is given in the first twenty-four to thirty-six hours after the initial chill. *b.* Many writers attribute favorable effects to the use of mercury (as calomel internally, externally as inunction

with unguent. hydrarg.). *c.* Inhalations of ether and chloroform (two to six times a day until narcosis begins) are said to be indicated when severe pains and marked cyanosis are present.

### 9. *Interstitial Pneumonia and Retraction of the Lungs.*

**I. ETIOLOGY.**—Interstitial pneumonia gives rise to profound changes in the interlobular connective tissue. The disease may be acute or chronic, the former variety exhibiting a tendency to the formation of pus, the latter to inflammatory hyperplasia of the connective tissue. Both forms occur rarely as an independent disease, but are usually of a secondary nature.

Acute interstitial pneumonia possesses merely an anatomical interest. It is a rare disease, and cannot be diagnosed during life. When fully developed, the interlobular connective tissue is found to be broken down into pus; the individual lobules, and even the infundibula, are separated from one another. The process may be confined to small spots or extend over a large part of the lung. The starting-point of the process is either at the surface of the lungs or the disease begins at the hilus, and spreads along the peribronchial tissue into the interlobular fibrous tissue (*pneumonia dissecans*).

The following are the causes of *pneumonia dissecans*: *a.* Fibrinous and catarrhal pneumonia, if the interstitial connective tissue, which is always infiltrated with round cells in these diseases, is affected too strongly. *b.* Infectious pulmonary emboli, which give rise to suppurative inflammation in their vicinity. *c.* Empyema. Rindfleisch states that the lymphatics of the lung are the means of spreading the inflammation, and that we can often follow, with the naked eye, the lymphatics, which are filled and surrounded by pus, from the surface of the lung into the interior. *d.* According to Hertina and Prevost, primary interstitial pneumonia sometimes follows injury or a cold, but an infectious factor is also necessary; moreover, it always occurs in exhausted individuals.

The symptoms of *pneumonia dissecans* are either concealed by the primary disease or a typhoid condition develops, the patients expectorate muco-purulent, purulent, or bloody masses, friction murmurs are heard (complication with pleurisy); in the beginning, we find the physical signs of infiltration of the lungs (especially dulness), and later, the signs of the formation of cavities. We have to deal, therefore, with a condition of purulent infiltration which is followed by perforation and expectoration of pus, and the formation of cavities. The differential diagnosis from pulmonary abscess is impossible.

Chronic interstitial pneumonia most frequently follows chronic bronchitis and pleurisy, but it may also be secondary to fibrinous and catarrhal pneumonia, pulmonary abscess, gangrene, phthisis, miliary tuberculosis, tumors and echinococci, pneumonokoniosis. The interstitial pulmonary changes following syphilis will be discussed in Vol. IV.

Primary interstitial pneumonia is rare, and is most apt to occur in old age. Some authors attribute it to malarial influences, others regard it, in certain cases, as a senile proliferation of connective tissue, such as develops not infrequently in the kidneys, liver, and even the heart muscle.

Chronic interstitial pneumonia is found at all ages. In children it follows measles and whooping-cough, which have given rise to bronchitis or catarrhal pneumonia. It sometimes develops quite rapidly, and ex-

tensive interstitial pneumonia may be found a few weeks after an attack of measles.

II. ANATOMICAL CHANGES.—Chronic interstitial pneumonia is either circumscribed or diffuse. In the former variety we find fibrous cicatrices or nodules which are often very firm, occasionally almost as hard as cartilage. The tissue is sometimes white (perhaps only in spots) or reddish-white in recent cases, sometimes it is gray or slaty, greenish-black or bluish-black from the presence of pigment. According to Rindfleisch, this is derived mainly from extravasations of blood. In diffuse interstitial pneumonia, the interstitial connective tissue appears as white, gray, or blackish stripes between the lobules. The alveoli are destroyed in part, and replaced by connective tissue. If retraction of the connective tissue occurs in peripheral portions of the lung, the surface becomes uneven and nodular (cirrhosis of the lungs).

The more the retraction of the connective tissue spreads, the more the volume of the lung diminishes. Circumscribed and diffuse interstitial pneumonia are often associated with another.

Since chronic interstitial pneumonia is generally a secondary condition, the primary pulmonary disease is, at the same time, also noticeable. The pleura is very often thickened to a greater or less extent, but it must be remembered that pleuritic thickenings and adhesions may develop secondarily in the rare cases of primary cirrhosis of the lungs. In other cases, the mucous membrane of the bronchi is found in a chronic inflammatory condition, and the bronchi may be dilated in places. The bronchiectasis may also develop secondarily in the cicatrix-like tissue, partly as the result of traction exercised by the retracting tissue, partly because the air is distributed very unequally during respiration, if a part of the pulmonary tissue has been destroyed and certain bronchi have undergone dilatation as the result of an excessive supply of air. In pulmonary abscess, gangrene, caseation, phthisical cavities, neoplasms, and echinococci, interstitial pneumonia gives rise to the formation of a firm, fibrous capsule, which, to a certain extent, protects the healthy tissue. If the lesion is the result of pneumokoniosis, the lungs are found filled with pigment (coal, iron, ultramarine dust, etc.).

The transition of fibrinous pneumonia into chronic interstitial pneumonia occurs not infrequently. According to Marchand, the first change is the development of young connective tissue in the alveolar spaces, and the interstitial proliferation of connective tissue occupies, to a certain extent, a subordinate place. The intra-alveolar young connective tissue is vascularized by new-formed vessels which pass from the capillaries of the alveolar walls into the new tissue. Marchiafava arrives at similar conclusions; he also observed proliferation of connective tissue in the fine bronchi. Advanced age, previous attacks of pneumonia, and, according to some, exposure during the attacks of pneumonia, are said to favor the transition into retraction of the lungs.

In chronic bronchitis and broncho-pneumonia, it is probable that the lymphatic vessels convey the inflammation-producers to the interlobular connective tissue, in which they give rise to slow inflammatory processes.

The lymphatics of the pleura maintain still more intimate relations with those of the interlobular connective tissue, so that it is not astonishing that pleurisy often gives rise to interstitial pneumonia. The most extensive interstitial changes are found in chronic and subacute pleuritis.



In pneumonokoniosis, the inhaled dust is in part deposited free in the interstitial tissue along the lymphatics, in part in amœboid cells which have passed from the alveoli into the interstitial tissue. But interstitial pneumonia appears to be the result rather of the accompanying chronic bronchitis than of the irritation from the dust.

III. SYMPTOMS.—Chronic interstitial pneumonia cannot be recognized until its sequel, retraction of the lung, has developed. It follows that the beginning of the inflammation cannot be diagnosed, especially as adjacent parts of the lung endeavor, by vicarious emphysema, to assume the function of those parts which are excluded from respiration.

The signs of retraction of the lungs may be circumscribed or unilateral. The most frequent circumscribed retractions are those which occur in the supraclavicular, infraclavicular, or supraspinous fossæ, associated with phthisical changes at the apices. Total unilateral retraction occurs most frequently after extensive pleurisy.

On inspection, the thorax is found to be depressed over the affected part. If the whole lung is involved, the entire side of the thorax is diminished in size. The thorax appears narrower, the intercostal spaces are smaller, the nipple is nearer to the median line, the thoracic muscles are less developed, the shoulder is lower on the diseased side. The spine is convex towards the healthy side, and the lower angle of the scapula is separated from the thorax.

The respiratory movements of the affected side are diminished, and occasionally almost entirely abolished.

Abnormal pulsations are sometimes very distinct. If the anterior median border of the left lung is drawn to the outside, on account of the retraction, the pulsations of the heart are strikingly distinct and diffuse, because an unusually large part of its anterior surface is in contact with the chest-walls. If the retraction has given rise to a diminution of the vertical diameter of the left lung, the apex beat appears in the fourth left intercostal space, sometimes higher. Not infrequently it is also displaced to the side, so that it is seen pulsating in the middle axillary line (diminished transverse diameter of the left lung). In the latter event, there is not infrequently a systolic protrusion in the second left intercostal space, which is followed by a feeble, short, and more diffuse pulsation during diastole. This corresponds to the systolic filling of the pulmonary artery, and to the diastolic unfolding of its semilunar valves, the force of which is often increased. But these phenomena are not observed immediately adjacent to the left border of the sternum (the normal position of the beginning of the pulmonary artery), but are about four to eight cm. distant from it, *i. e.*, the pulmonary artery is also displaced.

In total retraction of the right lung, the apex beat is sometimes not visible, because it is covered by the left lung which has undergone vicarious emphysema. The heart is sometimes drawn so strongly into the right thorax that its movements are visible in the fourth right intercostal space.

Palpation confirms the results of inspection. Measurement shows the amount of retraction. In the case illustrated in Fig. 79, the difference amounts to three cm; in reality, five cm. if we take into consideration the fact that the circumference of the right thorax is normally two cm. greater than that of the left. The pneumatometric valves and the vital capacity are so much more diminished the more extensive the inter-

stitial pneumonia, and the more the dilatation of the lungs is prevented by pleuritic adhesions.

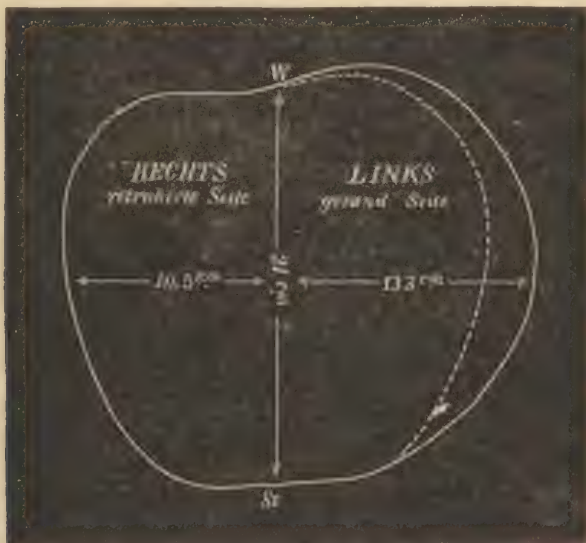
If large alveolar districts are deprived of air in consequence of the interstitial pneumonia, and large, permeable bronchi enter the non-aërated district, vocal fremitus will be increased, particularly if bronchiectasis or other cavernous formations are present. But if the bronchi are narrowed or obliterated by the interstitial process, vocal fremitus may be diminished or abolished.

Palpation often plays a prominent part in ascertaining the cardiac changes which have been mentioned above. The finger passed into the intercostal spaces often detects a pulsation which is not perceptible to the eye. This is particularly true of the diastolic pulsation of the pulmonary semilunar valves, and which is felt as a short, clicking blow alternating with the apex beat.

On percussion we find dulness, displacement of the boundaries of the lung, and immobility of its borders.

The dulness is owing in part to the absence or diminution of air in

FIG. 79.



Cyrtometer curve in retraction of the right side of the thorax, following pleurisy (at the level of the fifth costal cartilage). The dotted line on the left side shows the difference in size.  
 $\frac{1}{4}$  natural size.

the affected parts, in part to the narrowing of the intercostal spaces. If the affected parts inclose cavities, the dulness may be associated with a tympanitic or metallic quality, change of pitch, and other signs of cavity. In addition, percussion gives a feeling of increased resistance.

If the vertical diameter of the lungs is diminished, the apex will be found lower than normal. This is an extremely important sign of incipient phthisis. When the retraction is total, the lower border of the lung is also displaced upwards. On the right side, the upper boundary of the liver is unusually high (normal beginning of absolute [lesser] liver dulness in the right mammary line, between the sixth and seventh ribs), on the left side Traube's semilunar space becomes enormously high

(upper boundary normally is immediately below the apex beat, *i. e.*, immediately below the sixth rib). Since pleural adhesions are usually present, the respiratory movements of the edges of the lungs are absent or diminished.

In retraction of the left lung, a zone of loud pulmonary resonance is found not infrequently to the left of the edge of the sternum, and from the second to fourth costal cartilages. This is the result of vicarious emphysema of the right lung and the displacement of the mediastinum to the left by its anterior median border.

When the retraction is circumscribed or confined to one lobe, vicarious emphysema of the remainder of the lung often causes the pulmonary retraction to appear less than it really is.

The auscultatory phenomena depend upon the anatomical conditions. No respiratory murmur is heard over non-aërated cicatrices in which the bronchi and alveoli are occluded. If the bronchi are permeable, bronchial breathing may be heard. When the connective tissue incloses cavities, tinkling or metallic râles, metallic breathing, and metallic bronchophony may be heard. But schematization is impossible. Gross errors will be avoided only if we entertain clear ideas with regard to the genesis and physical significance of the phenomena.

Juergensen states that cirrhotic cracking occurs quite regularly in pulmonary retraction. He describes it as moderately fine, heard only at the height of inspiration, not changed on coughing, and intermediate between crackling râles and pleuritic friction sounds. It is produced probably in the catarrhal bronchi which are situated in the non-aërated parenchyma.

Auscultation of the heart possesses a certain degree of importance. The second diastolic (pulmonary) sound is intensified, corresponding to the palpable, often also visible closure of the semilunar valves.

The imperfect respiration is apt to produce attacks of frequent respiration, a feeling of want of breath, and cyanosis. On account of the vicarious emphysema which is produced, these symptoms may be very slight while the body is at rest, but they always appear during mental or physical excitement.

Retraction of the lungs also produces certain effects on the heart. If a certain portion of the pulmonary capillaries are destroyed, the blood pressure in the pulmonary artery must increase, *i. e.*, the right ventricle must undergo dilatation and hypertrophy.

Hence very serious disturbances may arise. If the power of the heart muscle diminishes, symptoms of stasis are the inevitable result. This is to be expected so much more readily in pulmonary cirrhosis, because the amount of work which devolves upon the right heart is unusually large. Not alone the destruction of pulmonary capillaries increases the amount of work to be performed by the heart, but the slight dilatability and impeded mobility of the lungs have an extremely important effect upon the circulation in the pulmonary artery.

IV. DIAGNOSIS.—We have previously stated that chronic interstitial pneumonia remains latent so long as there is no considerable diminution in the volume of the lung. The recognition of pulmonary cirrhosis is easy if we consider, not a single symptom, but the ensemble of symptoms. Among the individual symptoms, a pathognomonic value can be attributed only to partial or total retraction of the chest. Immobility of the borders of the lungs, increased elevation of the liver, displace-



ment of the heart, etc., may also be produced by other diseases (simple pleurisy, tympanites, etc.).

V. PROGNOSIS.—The prognosis can with difficulty be discussed from a general standpoint. It is so much more grave the more extensive the disease. Lesions of the valves or heart muscle render the prognosis more serious. In addition, the prognosis depends upon the primary affection.

VI. TREATMENT.—The treatment must be confined to general therapeutic directions. The patients should avoid bodily and mental exertion, take a light but nutritious diet, and avoid everything which requires increased labor on the part of the lungs or heart.

In many cases rational pulmonary gymnastics is advisable. The patients should take deep respirations several times a day, the arm being strongly elevated upon the side corresponding to the affected lung, and the chest bent towards the sound side. In this manner the affected side is forced to take a more active part in the respiratory movements than by the use of compressed and rarefied air, which is distributed uniformly to both lungs.

### 10. *Pulmonary Abscess.*

I. ETIOLOGY.—Pulmonary abscess leads to destruction of the lung tissue. The disease is rare and most frequently attacks men in middle life.

The following are the causes of the disease: *a.* Fibrinous pneumonia, especially when it attacks feeble individuals or drinkers, when it is very extensive, is situated in the upper lobes, and is associated with pulmonary hemorrhage. Fibrinous inflammation occurring in emphysematous and indurated lungs also exhibits a tendency to abscess formation. *b.* Catarrhal pneumonia may also give rise to abscess. *c.* Embolism of the pulmonary artery. This is a not infrequent cause, puerperal diseases or pyæmic processes forming the most frequent starting-point of the disease. But not every embolus is followed by an abscess. If it does not possess infectious properties, it will give rise merely to an hemorrhagic infarct. *d.* Injury to the lung. *e.* Foreign bodies in the bronchi. These may sometimes remain in the bronchi for months before the first signs of pulmonary abscess make their appearance.

Under the etiological conditions mentioned, abscess of the lung has a tendency to run an acute course. According to Traube and Leyden, pulmonary abscess may also run a chronic course, assuming an acute character from time to time.

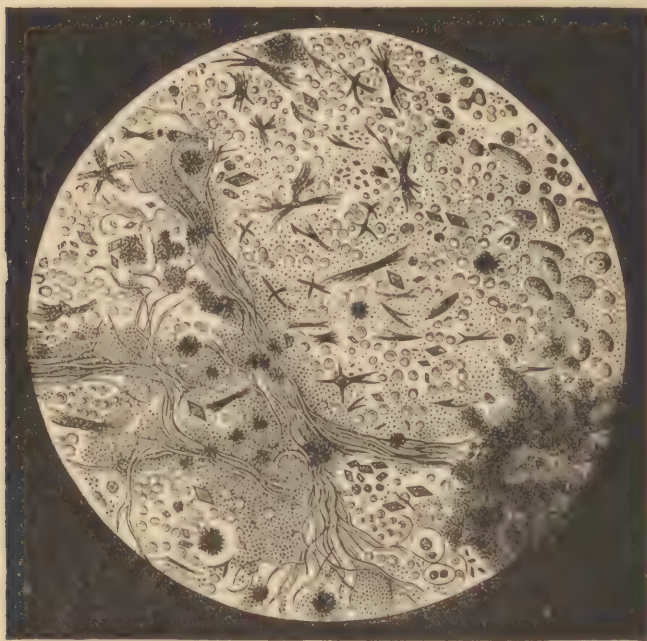
II. ANATOMICAL CHANGES.—In acute pulmonary abscess, a cavity filled with pus is found within the lungs. The size of the cavity varies from that of a pea to that of a large apple. Sometimes an entire lobe is occupied by the abscess cavity, and Beattie recently described a case in which, following a fibrinous pneumonia, almost the entire right lung had undergone suppuration. The cavity is sometimes irregularly round, sometimes jagged and chambered. Its inner surface is uneven, torn, villous, and in places (sometimes over the entire surface) is covered with a yellowish-green, gray, or brownish layer of pus. It is almost always surrounded by a fibrous capsule, which is the result of interstitial pneumonia around the abscess. Recovery is possible by means of retraction and the development of granulations on the inner surface. The contents of the cavity sometimes have a foul odor, but this is often a post-mortem phenomenon.

Pulmonary abscess occurs most frequently in the upper lobes. Several abscesses, particularly when embolic in their origin, are sometimes found. These abscesses are usually situated at the periphery of the lung.

III. SYMPTOMS.—The character of the sputum is an extremely important feature in the clinical history, and it alone renders the diagnosis possible. The sputum is purulent and almost always contains macroscopic shreds of the pulmonary parenchyma.

The daily amount of sputum is usually not inconsiderable; five hundred ccm. is not infrequent, and one thousand ccm. have also been observed. It generally has a stale, mouldy, sometimes buttermilk-like odor. If expectoration is impeded, the odor may become foul; but this disappears as soon as the sputum is again expectorated in larger quantities.

FIG. 80.



Sputum of pulmonary abscess. After Leyden.

If the expectoration is not free, the putrid decomposition of the pus is apt to lead to pulmonary gangrene. In typical cases, the sputum looks exactly like laudable pus. It is a homogeneous, greenish, opaque fluid, of creamy consistence and alkaline reaction. After standing, it separates into a lower, granular, sediment-like layer (mainly pus-corpuscles) and an upper serous layer. If the expectoration is very profuse, the uppermost layer may become frothy. In certain cases, the sputum retains a brownish color for a longer or shorter time, on account of the presence of a large amount of blood-pigment crystals. Occasionally the sputum is scanty, muco-purulent, lumpy, like that observed in phthisis.

Great importance attaches to the presence in the sputum of macroscopic shreds of the parenchyma. They form yellowish, yellowish-gray or greenish-gray, and smoky shreds which, on being shaken in water, are resolved into villous, shreddy, floating masses. Their number and size are sometimes astonishing, a diameter of three to six cm. being reached at times.

On microscopical examination, the sputum is found to be composed of the elastic fibres of the alveoli, mixed with cellular and crystalline elements and bacteria. In addition to mucous and pus-corpuscles, and desquamated epithelium, we notice fat crystals in the shape of radiating or tuft-like structures. Yellowish-brown or brownish-red pigment granules are also present. The abundance of hæmatoidin crystals is especi-

FIG. 81.



Sputum of chronic pulmonary abscess. Containing elastic fibres and tablets of cholestearin.  
After Leyden.

ally characteristic. They appear in the shape of well-developed plates, or of tufts which radiate in one or more directions and often contain pigment in their midst (Fig. 80). The occurrence of black pulmonary pigment is less important. Finally, we find bacteria, which are always round, usually of the same size and arranged in groups.

Shreds of parenchyma also appear in the sputum of chronic pulmonary abscess. They are generally black, but rarely present an alveolar structure. They consist generally of firm fibrous tissue; and, in addition to fatty mucous and pus-corpuscles and alveolar epithelium, contain cholestearin tablets.

The expectoration sometimes occurs in jets and mouthfuls. The



patient usually lies upon the affected side, so that a longer time elapses before the secretion reaches the mucous membrane of the efferent bronchus and gives rise to cough.

The physical signs of a cavity may be detected if the lesion is situated sufficiently near the surface. Remittent or hectic fever is often present; sweats and chills may also occur. As a rule, rapid emaciation follows.

The clinical development of pulmonary abscess varies according to its cause. If it follows fibrinous pneumonia, the crisis of the latter is usually delayed (beyond the fourteenth day of the disease), and is often incomplete. Fever soon reappears, the patients complain of difficulty of breathing and stitches in the chest, and relief is only obtained after the sudden expectoration of pus. Our suspicions should be aroused if the patient is feeble or addicted to drink, if the pneumonia is very extensive or the sputum is hemorrhagic. Traube showed that the grass-green sputum may alternate for one or two days with the hemorrhagic sputum. In one case Leyden found the first shreds of parenchyma on the sixteenth day after the beginning of the primary pneumonia. In another case of puerperal, embolic origin, the first shreds of parenchyma appeared on the twenty-third day after the occurrence of the pulmonary embolism.

The possible terminations of the disease are: *a.* Recovery, with the formation of a cicatrix and usually retraction of the lung. *b.* Incomplete recovery, the inner surface of the abscess cavity continuing to secrete purulent or muco-purulent masses. *c.* Death from hectic symptoms and increasing exhaustion, or suddenly from rupture of the abscess into the air passages. *d.* Transformation of the abscess into pulmonary gangrene by decomposition of the pus. *e.* Rupture of the abscess externally.

The rupture may give rise to pleurisy or pneumothorax, or the pus makes its way through the chest-walls, after previous adhesion of the pleural surfaces to one another. Senator has seen emphysema of the skin produced in this manner. Beatti noticed rupture of the abscess through the diaphragm, formation of an hepatic abscess, and finally perforation of the posterior chest-wall beneath the scapula. Pericarditis has been reported, in a number of cases, as a complication of pulmonary abscess.

IV. DIAGNOSIS.—The character of the sputum renders the diagnosis of pulmonary abscess easy. It may be mistaken for:

*a.* An abscess which has ruptured into the lung from the outside, for example, in empyema, cold abscess originating in the spine, hepatic abscess, pyo-pericarditis, suppuration of the bronchial glands, etc.

The differential diagnosis depends on the development of the disease, on the changes in other organs, and on the presence of shreds of pulmonary parenchyma in the sputum.

*b. Pulmonary Phthisis.*

In phthisis the macroscopic shreds of lung tissue and the hæmatoidin crystals are absent. At the same time the clinical history of the disease is different.

*c. Pulmonary Gangrene.*

The sputum of gangrene stinks and contains mycotic bronchial plugs. In the latter are found leptothrix pulmonalis and wavy needles of margaric acid. In addition, the number of hæmatoidin crystals is much less, and elastic tissue is present only exceptionally.

V. PROGNOSIS.—The prognosis is much more favorable in this disease than in pulmonary gangrene. Recovery is not infrequent, although the disease is always a serious one.

VI. TREATMENT.—The indications are to maintain the strength, limit the suppuration, and prevent decomposition of the pus.

We should order nutritious diet, alcoholic stimulants in generous amounts, quinine, iron, and cod-liver oil.

The second and third indications may be met at the same time. The air of the room should be kept pure and sprayed several times a day with carbolic acid (two per cent) or thymol (one per cent). We may also order inhalations of turpentine, carbolic acid, thymol, salicylate of soda, benzoate of soda, etc.

Surgical interference becomes necessary when perforation threatens. The opinion is gaining ground that the abscess should be opened even earlier, if we are sure that it is situated at the surface of the lung. Teale reports a favorable case. Paine a fatal one. In one case Finne observed the development of a fistula after the operation. When the case was reported, this fistula had lasted eight months.

### 11. *Pulmonary Gangrene.*

I. ETIOLOGY.—Pulmonary gangrene is mortification of the lung tissue, associated with processes of decomposition. It is a rather rare disease, occurring more frequently in males than in females, and developing generally between the ages of 16 and 40 years, though cases have been known to occur in early infancy and old age.

The disease occurs principally among the lower classes, inasmuch as its development is favored by poor nutrition, abuse of alcohol, living in damp and crowded apartments. It is said to occur at times in an epidemic form in poorly ventilated and crowded institutions. Pulmonary gangrene has also been observed frequently among the insane (melancholia with refusal of food, epileptics, drinkers). Some authors attribute this to overcrowding and poor ventilation of the asylums.

After severe infectious diseases (typhoid fever, measles, etc.) it sometimes develops after very slight causes, evidently because the organism is enfeebled and thus presents a more marked predisposition to mortification processes. We are not justified, however, in speaking of a primary predisposition (gangranæmia). It is well known, however, that marked gangranæmia develops in diabetes mellitus.

The following are the immediate causes of pulmonary gangrene:

a. Affections of the bronchi (bronchiectasis with putrid bronchitis and foreign bodies).

As the putrid process occurring within dilated bronchi is very similar to pulmonary gangrene, it is not surprising that the walls of the bronchi should occasionally become eroded and perforated, and the gangrenous process thus extend to the parenchyma. In like manner, foreign bodies may give rise to a putrid process in the bronchi, which extends later to the pulmonary tissue. In a case described by Greuser, gangrene occurred on the eighth day after a boy had swallowed an ear of rye five centimetres in length. On the twelfth day, the foreign body was expectorated, and complete recovery ensued. In carious processes in the ear, cancer of the lips, tongue, or tonsils, in noma, in operations on the mouth, putrid substances may enter the larynx, then pass deeper, and give rise to pulmonary gangrene. Trautvetter has recently described a case in which cerebral abscess is said to have given rise to pulmonary gangrene by means of embolism of the pulmonary artery. This case appears susceptible of more than one interpretation. Perhaps the cerebral abscess was

secondary to the gangrene of the lungs. Foreign bodies sometimes enter the respiratory tract after perforation of the air-passages, for example, in cancer of the œsophagus, vertebral caries, suppuration of the bronchial lymphatic glands, mediastinal suppuration and tumors, etc. Among the not infrequent foreign bodies are articles of food which "have gone the wrong way," especially in paralysis of the epiglottis, anæsthesia of the laryngeal mucous membrane, and in feeding by means of the œsophageal sound.

*b.* Diseases of the parenchyma of the lungs (inflammation, abscess, phthisis, and echinococci).

There can be no doubt that fibrinous pneumonia may pass directly into gangrene of the lungs. It must be remembered, however, that hepatization is not infrequently a result of preceding gangrene. Gangrene is most apt to develop after fibrinous pneumonia when the patients are feeble or addicted to drink, or when the infiltration is very extensive, so that the circulation becomes very much enfeebled or entirely interrupted, or if hemorrhages occur at the beginning of the pneumonia. An influence in this direction is also exerted, according to Rindfleisch, by the occurrence of putrid bronchitis in a pneumonic lung. Catarrhal pneumonia is a more infrequent cause of pulmonary gangrene. The connection between pulmonary abscess and gangrene is readily understood. Putrid decomposition of the contents of the abscess is alone necessary to cause gangrene. Traube explains its rare occurrence in phthisical cavities by the greater dryness of the latter. In one case, I observed gangrene as the result of echinococcus of the lungs. The vesicle was expectorated, and gangrene developed in the cavity thus formed.

*c.* Diseases of the pulmonary vessels (embolic gangrene).

Pulmonary gangrene will necessarily develop whenever the circulation in any part of the lung is interrupted. This is rarely the effect of hemorrhagic infarction produced by rupture of the blood-vessels, embolism, or venous thrombosis. In the English literature of the subject several cases are reported in which pulmonary gangrene is said to have resulted from falling into the water.

In embolic pulmonary gangrene, the emboli generally take their origin at the periphery of the body, are impregnated with septic substances, and, therefore, give rise to inflammation and putrid decomposition of the lungs. This may take place in marantic thrombosis, in decubitus, diphtheritic changes of the skin (in children particularly in diphtheria of the vulva), puerperal diseases, abscess of the liver, caries of the petrous portion of the temporal bone, etc.

*d.* Injuries to the lungs.

Perforating and gunshot wounds, and contusions of the lungs, give rise not infrequently to gangrene. The same lesion may follow a fall or blow against the chest without external injury.

II. ANATOMICAL CHANGES.—Pulmonary gangrene may be circumscribed or diffuse, the former varying from the size of a bean to that of an apple, the latter having no sharp boundary, and involving an entire lobe or even an entire lung. The affected parts stink, but the foul odor is sometimes less offensive than during life.

Circumscribed gangrene occurs not infrequently in multiple foci, more often on the right side than on the left, most frequently in the lower lobe, less frequently in the middle, and least frequently in the upper lobe. It is bilateral in exceptional instances.



The gangrene is found usually at the periphery, more infrequently within the lung.

When the gangrene begins in the upper lobe, several foci develop at a later period, evidently because a portion of the expectorated ichor has flowed into the bronchus of the upper lobe and there produced secondary gangrene.

The first changes in circumscribed gangrene begin with the formation of a brownish-black or greenish-black scurf. Then the peripheral parts begin to soften, separate from the surrounding tissues, and are situated like a sequestrum in a cavity filled with ichorous fluid. If the cavity is connected with a bronchus, the solid parts may be expectorated, or they first undergo complete softening and are expectorated gradually in a fluid condition.

The gangrenous cavity is usually of an irregularly rounded shape, uneven upon its inner surface, eroded, and filled with a stinking, grayish-green, crumbly, flocculent fluid. The bronchi usually empty into it with sharply-cut ends, and are in a condition of intense catarrhal inflammation. While these gangrenous changes are developing, the adjacent vessels are generally undergoing obliteration. It is only when the gangrene spreads very rapidly that patent vessels are opened, and a very free hemorrhage results.

The irritant action of the gangrenous ichor may give rise to loss of substance in the mucous membrane of the bronchi. Putrid bronchitis and bronchiectasis may also develop as the result of pulmonary gangrene.

The periphery of the cavity is often surrounded by a sort of fibrous capsule, which has developed as the result of interstitial pneumonia. This offers a possibility of the recovery of the process, the parts shrinking more and more, and the cavity undergoing gradual closure by the formation of granulations upon its inner surface. In other cases recovery is incomplete. The putrid decomposition of the contents of the cavity ceases, but the inner surface assumes the characteristics of a pyogenic membrane and constantly furnishes a purulent secretion. Moreover, the secretion may again become putrescent.

If the connective tissue capsule is wanting, the gangrene occasionally continues to spread and becomes diffuse. The adjacent portions of the lung are occasionally found to be œdematous or in a condition of hepatisation.

If the gangrene is situated near the surface, the pleura may become involved. Purulent or putrid pleuritis develops through the agency of the lymphatic channels, or the gangrene extends to the pleura and produces pleurisy directly, or the gangrenous focus ruptures into the pleural cavity, giving rise to purulent or ichorous pleurisy or pyo-pneumothorax.

Previous pleuritic adhesions may prevent perforation into the pleural cavity. In this way the gangrenous masses sometimes perforate the chest-walls and appear under the skin or externally. Stokes observed a case in which the ichor extended below the skin as far as the scrotum. Halley reported a case in which ichor and gangrenous lung tissue made their appearance below the mamma; the gangrenous mass was removed with the knife and recovery took place. Perforation of the skin may be preceded by emphysema.

Perforation sometimes takes place through the diaphragm or into the œsophagus and mediastinum. Rupture into the bronchi or trachea

is less frequent. Hertz describes a case in which gangrene extended to the costal pleura, diaphragm, peritoneum, and spleen.

In diffuse pulmonary gangrene, the tissue of the lung is converted into a blackish-green or grayish-green mass which has a nauseous odor, is permeated with ichor, and appears brittle and macerated. It passes gradually into the surrounding healthy tissue and exhibits a marked tendency to spread further. It is more frequent on the right side than on the left, and in the upper than in the lower lobes. The bronchial glands are generally swollen, sometimes even gangrenous.

III. SYMPTOMS.—Gangrene of the lungs may be entirely latent during life. The disease can be recognized with certainty only when the gangrenous spot communicates with the bronchi. Our suspicions should be aroused, however, whenever an individual, in whom the assumption of a pulmonary affection is warranted, suddenly begins to suffer from

FIG. 82.



Shreds of lung tissue from the sputum of pulmonary gangrene. Enlarged 275 times.

remittent fever, rapidly grows feeble, suffers from sweats and chills, but especially if the breath stinks, or the patient himself complains of a nauseous taste in the mouth or smells a cadaverous odor.

The character of the sputum is the most important symptom of manifest pulmonary gangrene. The sputum stinks and contains shreds of lung tissue. In cases of central gangrene this is the sole symptom.

The smell of the sputum is sometimes nauseous biting, like horseradish or garlic, sometimes peculiarly cadaverous and sweetish. It is communicated very rapidly to the surrounding atmosphere, so that the patient must sometimes be isolated. After standing, the smell of the sputum diminishes, and is sometimes lost in a few minutes. If the sputum is shaken the smell again becomes evident. The amount of the

sputum is sometimes but little over one hundred ccm., but generally it is more copious and may reach one thousand ccm. Expectoration is sometimes by the mouthful, and the sputum at times gushes from the mouth and nose.

It is always alkaline, but soon becomes acid on standing. After standing quietly, it separates into three layers. The upper one consists chiefly of froth, mixed with a few grayish-yellow or grayish-green clumps of pus and mucus. The middle layer is an ashen-gray or greenish-gray serous fluid which contains a few flocculi. The lower layer is granular, sediment-like, and contains the shreds of parenchyma.

FIG. 83.



Needles of fatty acids, from the sputum of pulmonary gangrene, mixed with drops of fat and pulmonary pigment. Enlarged 275 times.

These form black or blackish-gray masses, varying from the size of a pin's head to almost the size of a nail. They float in water and have a torn, villous surface. They consist of a transparent, colorless basement substance, which allows the recognition of the alveolar framework of the lungs and rarely contains elastic fibres (Fig. 82). In places we find yellowish drops of fat, clumps of black pulmonary pigment, and needles of the fatty acids. In addition, they contain a granular mass which, under sufficiently high powers, is found to consist of leptothrix pulmonalis.

The lower layer of the sputum also contains peculiar plug-like structures (mycotic bronchial plugs), which vary in size from that of a grain of sago to that of a bean. They are whitish-gray, or bright-brown, of the consistence of porridge, and on being squeezed emit a peculiarly bad smell. They consist of needles of the fatty acids (Fig. 83), yellow or brown flakes of pigment, hæmatoidin crystals, drops of fat, occasionally of more or less well preserved red blood-globules; the



chief constituent is an apparently granular detritus. Under sufficiently high powers, the latter is resolved into definite forms of fungi. These consist in the main of round granules and rods. Sometimes a number of granules are arranged in chains of greater or less length, or there are extremely long rods or rods with numerous members (Fig. 84 *a*). These structures have a lively movement. On the addition of tincture of iodine they assume a brownish-yellow, violet-blue, purple-violet, or blue color, but the contents of the individual elements, not the living membrane, are alone stained. Jaffé obtained from the plugs a snow-white substance which turned blue on the addition of iodine, but was neither an albuminoid, nor could it be converted into sugar by the action of saliva. Jaffé and Leyden applied the term *leptothrix pulmonalis* to this fungus, whose shape and reaction are the same as those of *leptothrix buccalis*, and attribute to it the power of producing putrefaction.

FIG. 84.



Microscopical constituents of the mykotic bronchial casts. After Jaffé and Leyden. *a*, *Leptothrix pulmonalis*; *b*, *cercomonas*; *c*, spirilli; *d*, eel-shaped threads of fungus; *e*, monas lens. Enlarged 800 times.

The same fungus made its appearance when the sputum of other diseases was allowed to putrefy. In addition to *leptothrix pulmonalis* the plugs and shreds of lung tissue contain spirilli (Fig. 84 *c*), and these are specially numerous when the sputum has a sweetish odor; also long, broad threads, with eel-like movements (Fig. 84 *d*). Kannenberg recently discovered in the sputum two forms of infusoria, viz., *monas lens*, and *cercomonas*. The former is a pale globule, only a little smaller than a red blood-globule and provided with a whip-shaped, sinuous appendage. The *cercomonas* also possesses a simple or bifurcated whip-like end (Fig. 84 *b*), and in addition a sort of riveting disk. The movements cease soon after the sputum is expectorated, and at the end of

twenty-four hours both infusoria can be distinguished only after staining with methyl violet.

The mycotic bronchial plugs are sometimes absent in pulmonary gangrene, particularly if the cavity has a somewhat regularly rounded shape, and the mode of entrance of the main bronchus permits free and ready expectoration.

The sedimentary layer of the sputum also contains more or less degenerated red and white blood-globules, and crystals of ammonia-magnesian phosphates.

On chemical examination of the sputum, Jaffé and Leyden found that it contained volatile fatty acids, especially butyric and valerianic acids, usually ammonia and sulphuretted hydrogen, often leucin, tyrosin, and traces of glycerin. Filehne and Stolnikow discovered a ferment-like body similar to trypsin, and which probably destroys the elastic fibres in the shreds of parenchyma.

The disease is almost always associated with fever of a remittent type. This often subsides for a few days or weeks and then reappears. The apyrexial interval often corresponds to a period during which the sputum has diminished in amount, and the putrid odor has subsided. Chilly sensations and even decided chills are not infrequent; profuse sweats are also often observed. All these symptoms are the result of the absorption of ichor. The pulse is very much accelerated, small, and soft. The vital energies of the patient are rapidly lost, emaciation occurs, and the complexion becomes pale or gray. In rare cases the strength of the patient remains unimpaired for a long time. This is often noticed in putrid bronchitis, and in doubtful cases is useful in differential diagnosis.

No local changes will be found in the thorax in cases of central gangrene. When diffuse gangrene is situated peripherally, the physical signs will be those of pulmonary infiltration, while in circumscribed gangrene with formation of a cavity, cavernous signs will appear. The most positive sign of a cavity consists in the fact that the percussion sound over a certain district is dull before expectoration, and tympanitic after expectoration. In addition, the boundary of the area of dulness is sometimes displaced on change of position, on account of the movement of the fluid contained in the cavity. It should be stated that the patients generally assume lateral decubitus upon the affected side in order that the secretion may accumulate in the cavity, and its constant discharge into the bronchi prevented. According to the point of entrance of the bronchus, the patients assume an elevated or low position of the body.

Hæmoptysis is a not infrequent complication of pulmonary gangrene. This is sometimes the first symptom of the disease, or it occurs at an advanced stage after violent coughing, or it develops spontaneously from erosion of a vessel. It is dangerous on account of its profuseness, so that suffocation may occur from filling of the bronchi, or death may follow the excessive loss of blood.

The masses of blood sometimes have a peculiar black-red color, and occasionally the microscope shows very few well-preserved red blood-globules.

Previous mention has been made of the complication with pleurisy and pneumothorax, and occasionally with gangrene of the integument. Complete anorexia, vomiting, and diarrhoea sometimes make their appearance. These symptoms are usually the result of decomposition of the food from the ingestion of putrid sputum.

Metastatic suppuration may also occur. Meyer reports a case of putrid cerebral abscess and hepatic abscess.

Lombroso reports that he has observed a gangrenous odor and leucin in the urine in pulmonary gangrene.

The disease may run an extremely rapid course. Diffuse gangrene sometimes leads to a fatal termination within a few days. In circumscribed gangrene the disease may extend over many weeks or months. Recovery may be complete or incomplete. In complete recovery the sputum loses its stench, becomes scanty, at first purulent, then mucopurulent, and finally ceases. These symptoms correspond to the formation of a fibrous cicatrix at the site of the previous cavity. If a cavity with smooth inner surface persists, the expectoration of purulent fluid will continue, and it is possible that the process of putrefaction will again appear.

If circumscribed gangrene shows no tendency to recovery, a sort of typhoid condition (fever, clouded sensorium, delirium, muscular twitchings, fuliginous coating on the lips and tongue) develops towards the close of life.

IV. DIAGNOSIS.—In many cases the diagnosis of pulmonary gangrene is easy, on account of the character of the sputum. It is most readily mistaken for putrid bronchitis, but in the latter disease the sputum contains no shreds of lung tissue, although a few elastic fibres may be present if the process of decomposition attacks the walls of the bronchi.

In emphysema which has ruptured into the lung, the expectorated masses sometimes have a nauseous odor, but the sputum is purulent and contains no bronchial plugs. The sputum of phthisis may also have a foul odor if the patients are so feeble that they are unable to expectorate, and the stagnant secretion undergoes putrid decomposition. This is an unfavorable sign, which forebodes impending death from collapse. The previous history will obviate any difficulty in the diagnosis between gangrene and phthisis.

If sputum is wanting, the diagnosis must usually be left open. A probable diagnosis may sometimes be made if the expired air has a cadaverous odor, but we should avoid mistaking this for *fœtor ex ore*. In the latter condition, the foul smell increases in intensity the nearer we approach the mouth of the patient, and is often lost at a short distance from him; but in pulmonary gangrene the stench is diffused with uniform intensity over a great area.

V. PROGNOSIS.—The prognosis of diffuse gangrene is bad. The process spreads uninterruptedly, and, as a rule, rapidly proves fatal.

Under proper treatment, recovery is not very rare in circumscribed gangrene. This will occur so much more probably the more vigorous and youthful the patient, the smaller the gangrenous spot, the less the tendency to destruction, and the more uncomplicated the condition.

VI. TREATMENT.—The patients should be kept constantly in a recumbent position because, on rising, putrid secretion is apt to pass into the lower air passages and thus to infect healthy portions of the lung. In addition to nutritious and easily digested food, the patients should receive large amounts of alcohol (brandy, strong wine, etc.).

If possible, they should be placed in a separate room, partly in order to prevent annoyance to those about them, partly in order that the atmosphere should be as pure as possible. In summer the room should be aired directly several times a day, at other seasons it should be aired through an adjoining room. It is also advisable to disinfect the



air by means of the spray (carbolic acid 2-4%, four to six times a day) or by vessels of hot water in which a few tablespoonfuls of turpentine have been placed.

The sputum should be expectorated into a vessel which contains disinfectants and may be tightly closed. Naphthalin (2.0 poured on the bottom of the spit-glass every morning), carbolic acid (5 per cent), permanganate of potassium (5-10 per cent), chloride of lime, or powdered charcoal may be employed.

In addition, the patient should wear an inhalation mask, and should inhale disinfectants as often and as long as possible.

The Curschmann mask consists of a round iron framework, the free border of which bears a rubber ring filled with air so that it will readily adapt itself to the shape of the face. By means of a rubber band, the apparatus, after being applied to the mouth and nose, is fastened around the head (Fig. 85). Anteriorly is a short tube, bearing an anterior and posterior wire framework, the former being easily removed from the tube. In this tube is placed a sponge soaked with disinfecting drugs, the wire framework preventing its falling out or coming in contact with the skin. The best remedy for inhalation is carbolic acid, beginning with a five-per-cent and increasing its strength to a fifty-per-cent solution—indeed, there is no objection to the employment of the pure acid. Turpentine is preferable, however, if the patients have a tendency to hæmoptysis. The patients soon grow accustomed to the inhalation. The feeling of oppression, which may be experienced at the beginning, soon disappears and many patients finally wear the mask night and day.

The employment of Siegle's inhalation apparatus (Fig. 32) is less effective. The substances used for inhalation are : carbolic acid (2-4 per cent), permanganate of potassium (0.1-0.5 per cent), boracic acid (2-4 per cent), benzoate of sodium (5-10 per cent), salicylic acid (0.2 per cent), thymol, infusion of chamomile, turpentine, aqua creasoti, balsams of Peru, Tolu, and copaiba. The inhalations should be repeated from four to six times a day.

The simplest mode of inhalation is the pouring of turpentine into a vessel of boiling water, and the inhalation of the vapor through a funnel held over the vessel.

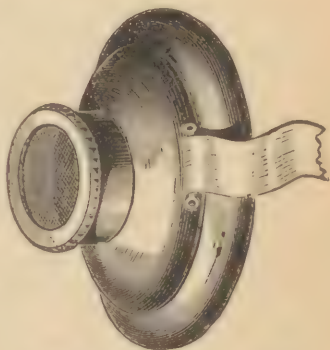
Stokes observed good effects from inhalations of chlorine; inhalations of bromine and oxygen have also been recommended.

Little can be expected from internal remedies. Traube recommended acetate of lead (0.05 every two hours) or, if no fever is present, tannic acid (0.3 every two hours) in order to diminish the amount of sputum.

Several attempts have been made to open the gangrenous foci and apply surgical treatment. Bull cured a case in twenty days, and Fenger reports another case of recovery; Carley and Smith met with no success. An operation appears specially indicated when the lesion is situated superficially. If several foci are present, Bull recommends that each be treated separately.

Special symptoms, such as violent cough, diarrhœa, etc., must be treated according to the principles already laid down.

FIG. 85.



Curschmann's inhalation mask.

## 12. *Neoplasms of the Lungs.*

1. Among the tumors of the lung, carcinoma and sarcoma alone possess clinical significance. The other forms of tumors (fibroma, lipoma, enchondroma, osteoma, dermoid cysts, cylindroma) usually give rise to very vague or no symptoms during life.

Carcinoma of the lung is primary or secondary, usually the latter. It occurs more frequently in men than in women, and usually from the age of twenty to thirty years, but exceptionally in childhood (Aldowie reported a case in a child of five and a half months). The causes of primary cancer are unknown. In certain cases injury has been mentioned as the immediate cause. According to Harting and Hesse an extraordinarily large number of the miners in the Schneeberger cobalt mines die of primary cancer of the lungs (75 per cent of all the deaths). The disease generally develops about the age of forty, after the patient has been working in the mines for upwards of twenty years; it must be regarded probably as the result of inhalation of arsenic.

Secondary cancer of the lung generally follows cancer of the breast, but may also follow cancer of any other organ. Sometimes there is a metastasis from remote organs, sometimes there is a direct propagation from adjacent parts (cancer of the thoracic walls, spine, œsophagus, mediastinum, etc.).

2. Soft medullary cancer is observed most frequently in the lungs, but alveolar cancer, scirrhous, and epithelial cancer have also been found. The carcinoma may be infiltrated or circumscribed. In the former variety there is a diffuse proliferation which passes into the healthy tissues without any sharp boundary; in the circumscribed form we find sharply defined nodules. In both cases the greater part of a lobe may be absorbed by the new growth. The tumor may attain the size of a child's head. In other cases there are innumerable small nodules resembling miliary tubercles (even the clinical history may be similar to that of miliary tuberculosis).

The cancerous nodules may be situated within the lung or at the surface, and often immediately underneath the pleura. In the latter event, they present not infrequently a central depression (cancerous umbilication). The cut section is generally white, soft, sometimes almost creamy; the tissue swells up above the cut surface, and is often traversed by fine injected vessels. The nodules are occasionally softened internally, and a part of the softened mass may be discharged through a bronchus during life, so that a cavity forms within the tumor.

In other cases, calcification and ossification develop within the cancerous nodules. Of twenty-seven cases of primary cancer of the lung collected by Reinhard, eighteen were found in the right lung, nine in the left lung. The right upper lobe is affected most frequently. The new-growth generally starts at the hilus, and then spreads to the periphery inside the walls of the bronchi, or around the bronchi, or along the adventitia of the vessels.

In many cases, cancer is also found in other organs. The bronchial, mediastinal, supraclavicular, infraclavicular, and axillary glands are often infiltrated, and may press upon the veins, giving rise to œdema and stasis of blood. Or the nerves may be compressed, and violent neuralgia produced. The growth may also proliferate directly into the nerves. As a result of the increased size of the affected lung, the heart may be displaced, and disturbances of circulation may arise in this

manner. The cancerous degeneration may also extend to the pericardium, heart muscle, and origin of the great vessels. Finally, pulmonary cancer may perforate the thoracic walls, and appear externally.

3. The symptoms of pulmonary cancer may be easily deduced theoretically. The diminution of the respiratory surface will produce disturbances of respiration (shortness of breath, cyanosis, irregular respiratory movements). If compression of the heart or venous trunks is produced, œdema and dilatation of the veins will result. In addition, we find a characteristic sputum, signs of absence of air in the pulmonary tissue, carcinomatous degeneration of peripheral lymphatic glands, occasionally metastases in peripheral organs. All these symptoms may be absent if the cancer attains only slight dimensions.

Death sometimes occurs suddenly from profuse hæmoptysis.

In certain cases, the symptoms are very vague: dyspnœa, asthmatic attacks, cyanosis, symptoms of bronchitis, a feeling of oppression, lancinating pains within the chest, mucous and not infrequently blood-stained sputum. The clinical history will sometimes warrant a probable diagnosis, especially if the symptoms develop after extirpation of a cancer; in other cases, the history resembles that of phthisis or miliary tuberculosis, especially if the patient suffers from profuse night sweats.

Among the objective changes we find dulness, which is often so irregular in shape that in doubtful cases this feature serves to differentiate the disease from pleurisy. If the lumen of the afferent bronchus is free, bronchial breathing is heard over the area of dulness. If the bronchus is occluded, neither respiratory murmur nor vocal fremitus will be found. The affected side of the thorax may be enlarged, and the heart displaced, if the cancer involves a large part of the lung. The cancer-mass sometimes makes its way to the outside through an intercostal space. Not infrequently we observe dilatation of the cutaneous veins, and œdema in the face, arms, and chest; these symptoms merit special attention if they appear only on one side. Hard, swollen glands near the clavicle and in the axilla should also be carefully considered.

Stokes was the first to call attention to the fact that the sputum occasionally presents a gelatinous reddish or blackish-brown appearance, evidently as the result of the intimate admixture of mucus and blood. But this form of sputum may also occur in other pulmonary affections. Elliot and Janssen described a grass-green sputum—the result of progressive transformation of the blood pigment. Cancer elements have been found in the sputum, and even pieces of cancer, which were visible to the naked eye, have been expectorated. In some cases the sole symptom of the disease is violent and repeated hæmoptysis. The sputum may acquire a very foul odor.

4. The disease may be mistaken for aneurism, if the pulse on the affected side is smaller than on the healthy side, and a systolic murmur is produced by compression of the aorta or pulmonary artery. Pulsation of cancerous masses lying over the heart may also lead to errors in diagnosis.

5. As a matter of course, the prognosis is unfavorable, and the treatment must be restricted to symptomatic measures.

6. The history of sarcoma of the lungs is the same as that of cancer, so that a differential diagnosis between the two is impossible during life. Kroenlein successfully removed from the lungs a sarcoma nodule, which was secondary to sarcoma of the chest-walls.



### 13. *Echinococcus of the Lungs.*

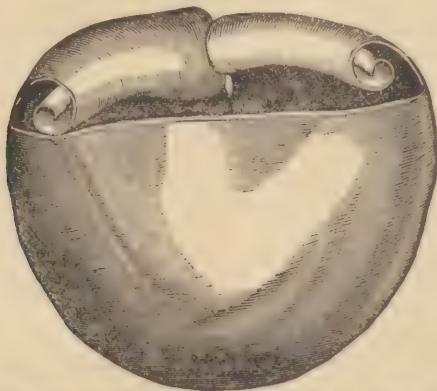
1. Echinococci may develop primarily in the lungs, or be conveyed from other organs. It is most frequently secondary to echinococcus of the liver. As a general thing, the vesicle breaks through the diaphragm, and then enters the lung; more rarely, the communication takes place through the hepatic veins, the inferior vena cava, and the right side of the heart. The lungs may also be affected secondary to echinococcus of the heart which has broken loose and been carried into the pulmonary artery.

Echinococcus of the lung is situated most frequently in the right lower lobe, more rarely in the upper lobes. It is occasionally found in several lobes, in exceptional cases in both lungs.

The size of the vesicles varies, but they may attain the dimensions of a man's head, or even occupy almost an entire lung. Under such circumstances, we find dilatation of the thorax, displacement of the heart, diaphragm, and liver, and, in young persons, curvature of the spine.

Scheuthauer recently described a case of multilocular echinococcus of

FIG. 86.



Expectorated echinococcus vesicle, with inrolled free edges. Natural size.

the lung, developing in the branches of the pulmonary artery. Andral found an echinococcus in the pulmonary vein.

Small vesicles may gradually calcify, and often remain for a long time in the lung as a corpus mortuum. In other cases suppuration occurs around the vesicle, which becomes loosened, passes into the bronchi, and is expectorated. In the most favorable event a cicatrix forms at the site of the vesicle, but suppuration and gangrene of the surrounding parts may also take place. As a rule, the vesicle is expectorated in little pieces, indeed, they may be so small as to be recognizable only under the microscope. The vesicle sometimes ruptures in consequence of bodily exertion, or violent coughing. If it is situated immediately beneath the pleura, it may rupture into the pleural cavity and into a bronchus at the same time, thus giving rise to pneumothorax.

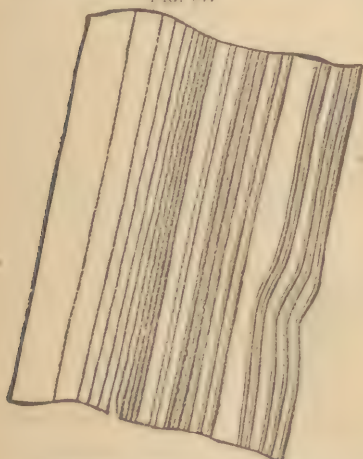
The echinococcus has also been known to rupture into adjacent organs; into the cavities of the pleura, pericardium, or peritoneum (with subsequent inflammation), into the stomach and intestines (followed by evacuation from the rectum).

2. Pulmonary echinococcus occurs in regions in which dogs are

numerous, the houses are not kept clean, and men and animals live together a good deal.

3. The disease can only be recognized with certainty if the vesicles, pieces of the vesicles, or their contents are expectorated. The vesicles and their shreds are readily recognized. They have a milk-glass or bluish-white color, are opaque, and the free edges of the larger pieces have a tendency to roll inwards (Fig. 86). Under the microscope they are found to be composed of parallel layers, which are separated from one another by slightly granular lines (Fig. 87). The contents of the vesicle may contain scolices and hooks (Fig. 88). If these parts of the echinococcus cannot be demonstrated in the sputum, the diagnosis, as a rule, is impossible. Death sometimes occurs unexpectedly from sudden rupture of a vesicle. In other cases recurring hemorrhages are observed for many months, and their cause is disclosed only at the autopsy. Sometimes pleurisy develops, remains stationary for a long time, and causes death from exhaustion. On autopsy, echinococci are found beneath the

FIG. 87.



Transverse section of an echinococcus membrane with parallel strata. Enlarged 275 diameters.

FIG. 88.



Echinococcus hooks. Enlarged 300 diameters.

pleura. If large echinococci are situated centrally, very vague respiratory disturbances are produced: a feeling of oppression, pain, dyspnoea, attacks of suffocation, occasionally fever and rapid emaciation. If an extensive echinococcus is situated immediately beneath the pleura, we will find the following local changes: dulness, abolition of the respiratory murmur and of vocal fremitus over the area of dulness, occasionally dilatation of some of the intercostal spaces, dilatation of the thorax, displacement of the heart and liver; if the compression of the lung is very marked, bronchial breathing may be heard at the periphery of the dulness. These symptoms change if the echinococcus vesicle is expectorated, and are replaced by cavernous signs (tympanitic or metallic tympanitic percussion sound, cracked-pot resonance, change of pitch on opening and closing the mouth, bronchial or metallo-bronchial breathing, increased bronchophony, consonant or metallic râles). The signs of a cavity will disappear and give place to those of non-aërated tissue when a cicatrix forms. In other cases the symptoms are followed by those of pulmonary gangrene.

Recovery is not unusual, if the echinococcus vesicles are expectorated. Very extensive and growing echinococci may prove fatal by causing suffocation. Death may also be the result of hæmoptysis, gangrene, rupture of the vesicle into the pleura, pericardium, or peritoneum, etc.

4. Treatment must be confined to maintaining the vital energies and combating prominent symptoms. Surgical treatment is necessary if the echinococcus ruptures externally. Fenger and Mosler successfully undertook the surgical removal of the parasite before rupture threatened. The attempt has also been made to cause the death of the echinococcus. Inhalations of ether, turpentine, benzin, natrium chloratum, salt-water baths, iodide of potassium, and mercurials internally, have been recommended for this purpose. If the vesicles have been expectorated, we should endeavor to prevent gangrene by inhalations of carbolic acid (2-4 per cent), thymol, etc.

#### APPENDIX.

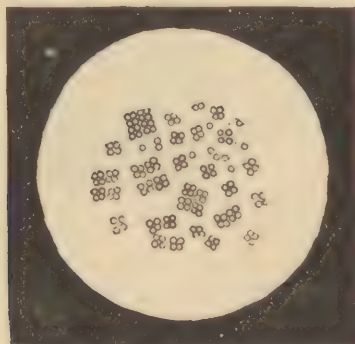
*Cysticercus cellulosæ* is also found in the lungs, but possesses merely anatomical interest. Diesing also reported a case in which large numbers of *strongylus longevaginatus* were present in the lungs.

#### 14. *Pneumonomycosis*.

Pneumonomycosis is the development of vegetable parasites in the lung (*schizomycetes*, *oidium albicans*, *aspergillus*).

*Schizomycetes* play a prominent part as a causal factor in many diseases of the air passages (putrid bronchitis, pulmonary gangrene and

FIG. 89.



Sputum in pneumonomycosis sarcinica. After Friedreich.

abscess). Heimer found sarcina in the sputum of a phthisical patient; they were in part free, in part inclosed in pus-corpuscles. Their abundant development appeared to be connected with the occurrence of a pneumonia. Friedreich also described a case of pneumonomycosis sarcinica (Fig. 89).

*Oidium albicans* was found by Rosenstein in a case of putrid bronchitis.

Pneumonomycosis aspergillica has been found mainly in the bodies of those who have died of chronic pulmonary diseases (phthisis, hemorrhagic infarction, gangrene, cancer). Fuerbringer found *aspergillus* in the sputum two days before the death of the patient. Rother describes a case of infiltration of the left apex in which grayish-green particles



were expectorated. Under the microscope they were found to be composed of necrotic pulmonary tissue infiltrated with aspergillus. The fungus disappeared in the course of a month, and the patient recovered.

## PART VI.

### DISEASES OF THE PLEURA.

#### 1. *Inflammation of the Pleura. Pleuritis (Pleurisy).*

I. ETIOLOGY.—Pleurisy is a very common disease. Fibrous adhesions between the pulmonary and costal pleura—the result of previous inflammation—are found in most bodies, even though no symptoms of pleurisy were observed during life.

The disease occurs at every age, and has been found even in the fœtus. Lawrence has recently maintained that idiopathic pleurisy is not infrequent in the new-born. During childhood it is found most frequently before the age of two years. The largest number of cases occur between the ages of twenty and fifty years. The predominance of the male sex is owing to the fact that it is more exposed to the causes of the disease.

Pleurisy may be either primary or secondary. In primary pleurisy a very prominent part is played by catching cold (pleuritis rheumatica).

It is produced not infrequently by a wetting and sudden cooling of the body. The danger of being affected with the disease is so much greater the less resistant the individual is. This explains the fact that patients who have passed through any protracted illness are apt to suffer from pleurisy, although they may not have left the sick-room.

Statistics show that pleurisy is more common in winter and spring than in the autumn and summer. Some observations appear to favor the view of the epidemic occurrence of the disease, but it should not be forgotten that injurious telluric influences usually affect large numbers of the community at the same time. Certain forms of pleurisy, particularly pleuritis acutissima, undoubtedly produce the impression of a severe infectious disease, but at the present time we are unable to sharply differentiate, among the group of rheumatic pleurisy, between purely rheumatic and infectious cases.

Injury also acts as a cause of primary pleurisy. This has been observed after a blow, fall, or concussion. Inflammation of the pleura may alone result, or the disease may be associated with traumatic changes in the thoracic muscles or bones, or in the lungs.

Secondary pleurisy occurs most frequently as the result of inflammation of adjacent organs.

Those cases which accompany inflammatory changes in the pulmonary parenchyma are the most frequent. The disease occurs almost constantly in fibrous pneumonia, but its secondary nature cannot always be proven. Perhaps the same cause which produced the pneumonia also gives rise to the pleurisy. This view is most probable in those cases in which the inflammation of the pleura predominates over that of the parenchyma of the lung. Pleurisy may also complicate catarrhal pneumonia, phthisis, pulmonary abscess, gangrene, hemorrhagic infarction, echinococci of the lung, bronchitis, and peri-bronchitis, though a direct

propagation of the inflammation does not obtain in all cases (propagation of the inflammation-producers through the lymph channels). Pleurisy always occurs when destructive processes in the lung cause destruction of the pulmonary pleura and perforation into the pleural cavity.

Inflammation of the pericardium, sternum, ribs, and spinal column may also be followed by pleurisy. In certain cases it is the result of cancer of the mammary gland and œsophagus, or abscess of the latter organ following the ingestion of foreign bodies. Inflammation of the cellular tissue of the neck or mediastinum extends occasionally to the pleura. Pleurisy may also be produced by inflammations within the abdominal cavity (peritonitis, abscess of the liver, spleen, kidneys, perinephritis, perityphlitic abscess, psoas abscess, etc.). The inflammation may spread through the agency of the numerous lymphatics of the diaphragm, or deposits of pus break through the diaphragm into the pleural cavity. Under such circumstances, the latter sometimes contains coloring matter of the bile, fæces, worms.

Pleurisy which develops in the neighborhood of an abscess is not necessarily purulent.

Profound changes in the blood and the metabolic processes are frequent causes of secondary pleurisy. We may mention Bright's disease, gout, scurvy, cachexiæ in general, and valvular lesions of the heart. In another series of cases the infectious diseases constitute the primary cause. In this category may be included measles, scarlatina, small-pox, acute articular rheumatism, diphtheria, ulcerative endocarditis, gonorrhœa, pyæmia, and septicæmia. Pleurisy occurs much more rarely in typhoid fever, and usually not until the fever has become remittent.

In all probability the bacteria, to which these infectious diseases owe their origin, exert a direct inflammatory irritation upon the pleura, to which they have been conveyed through the lymphatics. Certain epidemics of infectious diseases are characterized by their frequent combination with secondary pleurisy.

Finally, secondary pleurisy may be the result of diseases of the pleura itself (tuberculosis, carcinoma, sarcoma).

In the majority of cases, pleurisy is unilateral. If it is bilateral, the primary cause is generally tuberculosis, cancer, Bright's disease, gout, scurvy, syphilitic cachexia, in short a general disease.

II. ANATOMICAL CHANGES.—Pleurisy may be either dry or fluid. The former variety leads to the production of fibrinous deposits, the latter to the formation of an exudation, which may be serous, purulent, ichorous, or bloody, or a mixture of various forms.

In the beginning all forms of pleurisy are alike. The changes generally begin with unusual dilatation and hyperæmia of the subserous and serous vessels. The inflamed portion of the pleura thus assumes a very red appearance, which is sometimes uniform, sometimes in the form of closely aggregated streaks, sometimes in the form of islands and patches. In certain places we may find rupture of the vessels and small extravasations which extend occasionally to the free surface of the pleura.

These changes are followed very rapidly by swelling of the serous and subserous tissue, evidently as the result of exudation from the dilated vessels. The free surfaces of the pleura soon lose their lustre and have a dull look (swelling, cloudy swelling, and partial desquamation of the endothelium).

The inflammatory changes terminate temporarily in the production, upon the surface of the pleura, of cobweb-like or thick, firm membranes which are readily removed with the aid of a scalpel. These membranes are composed of exuded fibrin, whose origin Virchow attributes to the parenchyma of the pleura, while Rindfleisch believes that it is derived from the dilated blood-vessels and coagulates upon the free surface of the pleura. On microscopical examination of the membranes, we find a fibrillated basement substance, in which a larger or smaller number of white corpuscles are situated. On the addition of acetic acid, the basement substance swells and assumes a homogeneous appearance.

In dry pleurisy these changes alone are produced; they may be either circumscribed or diffuse. As a rule, the process affects both surfaces of the pleura on one side of the thorax, and is rarely confined to the pulmonary or costal pleura.

If a dry pleurisy resolves, the membrane is absorbed, its constituents undergoing partly mucous, partly fatty changes. In other cases the membranes organize into fibrous adhesions between the costal and pulmonary pleura, the round cells contained within them being converted partly into connective-tissue cells, partly into new blood-vessels by juxtaposition and hollowing out. Occasionally we find that, instead of adhesions, thickenings of the pleura are produced.

Sometimes an entire lobe or lung is adherent to the chest-walls by more or less firm connective tissue, sometimes there are ribbon-shaped adhesions, finally we may find villous appendages which are occasionally the result of rupture of previous band-shaped adhesions.

In fluid pleurisy, the first changes are exactly similar to those mentioned in the description of dry pleurisy, but they continue with greater or less rapidity until a fluid exudation accumulates in the pleural cavity. If the exudation is serous, it generally appears as a yellowish or yellowish-green fluid which is rarely perfectly clear, but is more often cloudy and mixed with flocculi. Cases of pure serous pleurisy are not frequent. As a rule, we find more or less firm, thick, yellow fibrinous coagula, partly in the fluid, partly upon the surface of the pleura. These accumulate generally upon those parts which take a relatively small part in the respiratory movements (the fissures between the lobes and the space between the diaphragm and the base of the lung). At those places at which the fibrinous masses upon the pulmonary and costal pleura come in immediate contact, their surface appears very uneven, like a network—an appearance which is evidently the result of the friction of the pleural surfaces against one another.

The fibrinous coagula consist of a fibrillated basement substance and a few round cells. The serous fluid usually contains but few cellular elements: round cells, desquamated endothelium, and a few red blood-globules. If the exudation has been formed a long time, the cells present signs of fatty degeneration, and the fluid is also found to contain free drops of fat.

In purulent pleurisy, the fluid exudation is similar to the pus of an ordinary abscess. It is an opaque, greenish or greenish-yellow fluid which separates after standing into two layers, the lower one consisting mainly of the cellular elements, the upper of the pus serum. Fibrinous coagula are rarely absent in this form of pleurisy; they are often extremely soft, and contain a large proportion of cellular elements. Purulent pleurisy may exist as such from the beginning, or it may develop



from a preceding serous pleurisy. The latter mode of origin is observed in the majority of cases.

A large proportion of the pus-cells are white blood-globules which have emigrated from the vessels of the pleura. It may be assumed, however, that the pus is also derived from other sources. In his experimental investigations, Rindfleisch noted their development from endothelium cells, the nuclei of which proliferated and then separated from the mother cell. It has also been maintained that the pus-cells may originate from the fixed connective-tissue cells of the subserous and serous tissue.

The microscope shows intact and fatty pus-cells in the purulent exudation. In one case I found crystals of a double pyramid shape, exactly similar to Leyden's asthma crystals.

In ichorous pleurisy, the fluid has a grayish-green or dirty reddish-brown color, and a biting, stinking, cadaverous odor.

On microscopical examination, we find in the main a granular detritus, composed in part of bacteria. In one of my cases the exudation contained an enormous number of needles of the fatty acids. In those cases which are the result of putrid bronchitis or pulmonary gangrene, the exudation contains peculiar brownish crumbs or plugs, consisting of margaric acid crystals, drops of fat, pulmonary pigment and leptothrix pulmonalis (vide page 325).

Hemorrhagic pleurisy is easily recognized by the bloody color of the fluid exudation. In recent cases, when a large amount of blood is mixed with the exudation, the fluid may look like freshly drawn blood, in older cases it has a brownish-red or brownish-black appearance. In the former event the red blood-globules are usually unchanged, in the latter they may be swollen, discolored, degenerated. White blood-globules and granulo-fatty cells are also present. The blood-globules are derived to a slight extent from extravasations, the majority passing into the pleural cavity by diapedesis from the blood-vessels.

I recently observed, in a typhoid fever patient, a hemorrhagic exudation which was lac colored and contained hæmoglobin; all the red blood-globules were destroyed.

The exudation sometimes contains a large amount of fat, and looks like chyle or milk; it contains drops of fat, fatty cells, and also cholestearin crystals. This is observed in cancerous pleurisy, but sometimes independently of cancer.

The amount of the pleuritic exudation may vary from a few tablespoonfuls to fifteen or twenty pounds, or even more.

The specific gravity of the fluid is generally from 1.015 to 1.023. According to Méhu, a specific gravity under 1.015 indicates that the fluid is the result of transudation, a specific gravity above 1.018 points to its pleuritic origin. The amount of albumin in serous exudations varies from 3.5-7 per cent. Gerhardt detected paralbumin in serous and purulent exudations. In many cases sugar is present, in others a glycogen-like body, which is converted by saliva into sugar. Naunyn constantly found urea, uric acid, and cholestearin. The latter was especially abundant in purulent exudations, after they had been exposed for some time to the air. Leucin, tyrosin, and xanthin were also derived from purulent exudations.

In serous exudations the amount of  $\text{CO}_2$  varies from forty to sixty-three per cent and increases with the duration of the exudation. In purulent exudations the amount of  $\text{CO}_2$  is so much less the greater the

number of pus-cells present. Traces of nitrogen and oxygen are also found.

The fluid is not always freely movable in the pleural cavity, because as a rule there are numerous adhesions between the costal and pulmonary pleura. The adhesions are often so extensive that they form a sort of wide-meshed sponge, the interstices of which are filled with fluid. Sometimes there is an adhesion of the layers of the pleura along the entire surface of the fluid (encapsulated pleurisy). As a rule, the encapsulation is a secondary change.

Large exudations do not find room in the pleural cavity unless the thoracic and abdominal viscera are displaced and the thorax is dilated.

The lungs are first affected by the accumulation of fluid. In the beginning they float upon the surface of the fluid, but when the latter becomes excessive, the lungs are compressed and converted into a firm, non-aërated tissue. The compression first occurs upwards and anteriorly, finally upwards and posteriorly. If the compression is complete, the lungs constitute a non-aërated, flattened structure, of a tough and leathery consistence and a grayish-red, brownish-red, or blackish color.

Next to the lungs the mediastinal organs, particularly the heart and large vessels, suffer displacement and compression. If the exudation is situated on the left side, the heart is displaced to the right, so that its right border may extend into the space between the right mammary line and the right axillary line. The heart is usually displaced to the right as a whole, so that the apex remains directed towards the left. Torsion of the heart, so that the apex is pushed furthest into the right thorax, may occur, but is much rarer. In right-sided pleurisy the heart is pushed further into the left thorax, so that the apex may be situated in the left axillary line. The heart is very often lower than normal.

The exudation also presses upon the diaphragm and the subjacent organs, the liver and spleen. The diaphragm is lower than normal, becomes more flattened or may even be convex towards the abdominal cavity. In right-sided pleurisy, the liver is pushed downwards; the right lobe is usually exceedingly low, while the left lobe is abnormally high, so that the liver has evidently been twisted.

Fraentzel showed that this torsion of the liver is not observed in all cases. In extensive pleuritic exudations, the diaphragm may be lower than normal on the healthy as well as the affected side. Hence the liver as a whole may be pushed downwards. If the depression is especially marked upon the affected side, the liver may be bent at an angle in its median line.

In pleurisy of the left side, the spleen is displaced. It is generally pushed downwards and towards the median line, so that it projects to a greater or less extent into the left hypochondrium. It is turned occasionally upon its long axis, so that the latter is no longer parallel to the course of the ribs. The stomach also undergoes a change in position, particularly at the fundus.

III. SYMPTOMS.—Not a few cases of pleurisy are attended with such slight symptoms that they remain latent during life. This is generally the case in dry pleurisy of slight extent, which can only be recognized by its sequelæ, as soon as adhesions impair the respiratory locomotion of the lung or the complementary pleural spaces are obliterated.

The objective symptoms are often restricted to local changes; at the most, the patient complains of pain in the side and disturbance of respiration.

In other cases, general symptoms are also noticed, the phenomena of fever being prominent among them. Sometimes the disease begins acutely with one or usually several chills followed by considerable, often continued fever. This disappears at the end of three or four weeks, the local changes in the thorax likewise undergoing resolution. Not infrequently the disease begins in a subacute manner. There are repeated chilly sensations at the outset, the fever is irregular and continues four, six, or eight weeks. Finally, there are chronic cases in which pyrexial and apyrexial periods alternate with one another. Cases have been reported in which the disease lasted upwards of twenty years.

The severity of the general symptoms does not always depend upon the local changes. In extensive pleurisy, the general symptoms may be slight, and vice versa. None of the general phenomena will enable us to make a positive diagnosis, so that the latter is rendered possible only by the local changes. This is especially true of secondary pleurisy, in which the general symptoms may be entirely concealed by the symptoms of the primary disease.

Pain and pleuritic friction murmur are among the chief symptoms of dry pleurisy. The diagnostic interpretation of the pain may admit of doubt, but the friction murmur is positive proof of the existence of dry pleurisy. In some cases, however, the friction murmur is permanently absent.

If the patients take to bed, they lie, as a rule, upon the healthy side.

This position is assumed because the slightest pressure upon the affected side increases the pleuritic pains. Traube also showed that decubitus on the affected side may cause stasis in the veins of the subserous and serous tissues, so that the adjacent nerves are irritated. However, decubitus upon the healthy side is not observed in all cases, the position depending chiefly on the irritability of the patient and the severity of the pain. We often find that at the beginning of the disease, when the pains are most intense, the patient lies constantly upon the healthy side, later he assumes the dorsal decubitus or may even lie upon the affected side.

The affected side of the thorax takes less part in the respiratory movements than the healthy side. At the same time the respiratory excursion of the thorax on the affected side usually begins later than on the healthy side, and it may even be interrupted and jerky.

This phenomenon is the result of the pleuritic pain. Every vigorous respiratory movement must increase the pain on account of tension of the pleura, and each incautiously-performed respiration will produce an involuntary arrest of the respiratory mechanism by the sudden pain. If the pains are very severe and diffused, the affected side may be almost motionless during respiration. If the pains are confined to an upper or lower half of the chest, it will sometimes be found that the unaffected half moves with unusual vigor.

A sort of temporary deformity of the thorax is sometimes observed. The shoulder of the affected side is depressed, the intercostal spaces are narrowed, the thorax seems to be drawn inward, the integument can be drawn into a fold more readily than upon the healthy side, and the spine is scoliotic with the convexity towards the healthy side. These changes are evidently the result of the spinal scoliosis, and are produced involuntarily in order that the inflamed costal pleura should be as free of tension as possible. When the patients assume complete dorsal decubitus these phenomena rapidly disappear.



Signs of cyanosis are noticed not infrequently upon the cheeks and visible mucous membranes; the more irregular and superficial the respirations the more marked is the cyanosis.

Pressure upon the intercostal spaces is more painful than that upon the ribs because the latter diminish the force of the pressure. Each intercostal space should be carefully palpated from in front backwards, and the boundaries of the painful region marked with a pencil. From the size of this region we may judge, to a certain extent, of the extent of the inflammation, although we know with regard to other inflammatory processes that the region of pain does not extend to the boundary of inflammation at all points, while at others it passes beyond the inflammation. As a rule, the boundaries marked upon the thorax are irregular in shape and extend over more than one intercostal space. They are found most frequently upon the lower, anterior, and lateral portions of the chest, and the parts adjacent to the nipple are apt to be especially sensitive.

If a pleuritic friction murmur of considerable intensity is present, this is sometimes conveyed to the hand as pleural fremitus. This is felt sometimes as a gentle stroke similar to that felt on passing the finger rapidly over silk, sometimes as a creaking, crackling sensation, as if a snow-ball is squeezed between the hands, or a stiff piece of sole leather is bent to and fro.

As a general thing, the leathery friction fremitus is not continuous, but is distinctly interrupted. It is sometimes felt only at the height of inspiration, in other cases it is felt during inspiration and expiration, in the rarest cases it is heard only during expiration. As a rule the impression is produced as if two rough surfaces were rubbed against one another from above downwards or in the opposite direction. The friction fremitus almost always increases if deep inspirations are taken. Its intensity is also increased at times by firm pressure upon the intercostal spaces. It is often very temporary and may disappear permanently after a few minutes or hours. In other cases it disappears after a patient has taken a few deep respirations, but soon reappears. Occasionally it lasts for weeks, months, or even years.

Vocal fremitus is unchanged in dry pleurisy, since the thin membranous deposits which cover the pleura are unable to affect it materially. Nothing abnormal is found on percussion.

The respiratory murmur is often very feeble and interrupted upon the affected side, on account of the diminished vigor and jerky character of the respiratory movements. But the chief symptom of dry pleurisy is the pleuritic friction sound. This is sometimes a superficial, gentle stroking, sometimes a loud, creaking or crackling sound. It may be interrupted and jerky in character. It is sometimes so loud that it is audible to the patient himself, or is heard at a distance from him.

It was formerly held that the friction sound is not heard until after the pleurisy has lasted several days. But Lebert noticed it on the first and second days of the disease, and Fraentzel claims that it is audible within twelve to fourteen hours. A deposit upon both layers of the pleura is not necessary to its production. Kuessner and Ferber found the friction murmur when the inflammation was confined to a single layer of the pleura.

The pleuritic friction sound is heard most frequently at the height of inspiration, much more rarely at the beginning. Sometimes it is heard only during deep and accelerated respiratory movements. It is

heard occasionally both during inspiration and expiration, least frequently during expiration alone. If deep inspirations are taken for a considerable period, it may suddenly disappear for a longer or shorter interval, evidently because the rough surfaces of the pleura have become smoother, on account of the continued vigorous friction. It may also disappear spontaneously for days, and then reappear unexpectedly, without any apparent change in the local or general conditions.

The intensity of the murmur is not infrequently increased by vigorous pressure with the stethoscope, and in rare cases it is audible only when a certain amount of pressure is thus exercised. Its duration varies from a few minutes to several years. Wintrich reports a case in which it was heard for four years. It is heard most frequently over the anterior and lateral inferior portions of the thorax. When heard only over the apices, it rouses a suspicion of phthisical changes complicated by secondary pleurisy.

The sound may have a very peculiar rhythm if the inflammatory process has developed near the pericardium (more frequently in left than in right pleurisy). In such cases, the murmur is apparently dependent on the movements of the heart, so that it may be mistaken for a pericarditic friction sound. The differential diagnosis will be discussed later.

To a certain extent, the cough constitutes a transition from the local changes to the general symptoms. It is often a very annoying symptom, which is productive of great pain and loss of sleep.

Some authors maintain that pleurisy as such does not give rise to cough, but that this is the result of a complicating bronchitis. Experiments on the production of cough by mechanical irritation of the pleura do not agree in their results. My own opinion is that inflammatory irritation of the pleura produces violent cough. It will be found that many of the patients cough without expectorating or presenting other evidences of bronchitis. In many healthy individuals I have been able to provoke cough by gentle pressure in an intercostal space. In cases of empyema which have undergone operation, I have also been able to produce cough by mechanical irritation of the pleura.

Stitches in the side are observed very frequently in dry pleurisy, independently of cough. When these pains are absent, the disease is recognizable only from the presence of the pleuritic friction sound.

In some cases the pain is felt upon the healthy side alone. Gerhardt explains this fact by the assumption of anastomoses in the mediastinum between the thoracic nerves of both sides, but Huss has been unable to demonstrate them anatomically.

The disease often begins suddenly with febrile symptoms. The scene opens with a chill or prolonged chilly sensations, and this is followed by continued, subcontinued, or irregular increase of temperature. This is associated with other febrile symptoms, viz., acceleration of the pulse, increased thirst, general malaise, scanty diuresis.

Dry pleurisy often exists as an independent disease, or it is a preparatory, complicating, or secondary affection in pleurisy with effusion. The latter disease generally begins as a dry pleurisy. If the fluid exudation is afterwards absorbed, the rough surfaces of the pleura come in contact with one another, and the conditions of dry pleurisy again develop. Finally, fibrinous deposits and adhesions are often found at the surface of an inflammatory exudation, so that the signs of fluid are associated with those of dry pleurisy.

Pleuritic adhesions form the chief sequel of dry pleurisy. They may give rise to obliteration of the complementary pleural spaces, or to fixation of the borders of the lungs. In these conditions, the respiratory movements of the lower and median borders of the lungs cannot be demonstrated on percussion.

Riegel and Tuczek observed inspiratory intensification of the apex beat of the heart when adhesions passed from the anterior border of the lung to the outer surface of the pericardium. As the fibrous adhesions are drawn upon during the inspiratory increase in the volume of the lungs, they cause the heart to approach the chest-walls more closely, and thus make the apex beat more distinct during inspiration. I have also observed this symptom in other conditions, viz., in a case of diffuse bronchitis which affected chiefly the anterior inferior portions of the lungs. At each inspiration the intercostal spaces were drawn in deeply; and as the lungs were only slightly movable on account of the bronchitis, the apex of the heart was closely approximated to the chest-walls during inspiration, and the apex beat thus became more distinct during inspiration. The symptom disappeared entirely after the bronchitis subsided.

The pleuritic adhesions are sometimes so complete that one or both lungs are firmly fixed over their entire extent. But as the mobility of the lungs exerts a great influence on the circulation, especially on the emptying of the pulmonary artery, the circulatory apparatus necessarily becomes affected. At first the right ventricle is alone affected, but the disturbance is soon conveyed to the left ventricle, and finally the entire heart undergoes hypertrophy and dilatation. Phenomena of stasis are apt to develop under such circumstances, particularly in the lungs, on account of the impaired mobility of these organs. Emphysematous changes develop not infrequently even when the pleuritic adhesions are circumscribed. Emphysema may develop beneath the adherent parts (produced by unequal dilatation of the lungs, and unequal distribution of air in them).

In fluid pleurisy the chief interest also attaches to the local changes. Percussion offers the most important diagnostic aid. The presence of fluid in the pleural cavity will not be recognized if dulness is absent, and an accumulation of fluid below a certain amount remains latent during life. Ferber found that it was necessary to introduce one hundred and twenty ccm. of water into the chest of the body of a child of twelve years to produce feeble dulness a finger's breadth over the posterior inferior surface of the chest. In an adult, four hundred ccm. were necessary to produce dulness of about two fingers' breadths. In the adult, about one-half litre of fluid must be present to permit of a positive diagnosis.

As a general thing the exudation must be two cm. in thickness in order to produce dulness; even then care is necessary, since the dulness will be distinct on gentle percussion alone.

The local changes depend almost exclusively on the amount of exudation.

Among the symptoms observed on inspection, the position of the patient often attracts attention. Patients suffering from pleurisy with effusion constantly maintain a position upon the affected side. If a dry pleurisy becomes fluid, the change is recognized by the change in the position of the patient from the sound to the diseased side.

If the patient lies upon the affected side, the healthy side of the thorax is directed upwards, so that its respiratory movements are the freest possible.

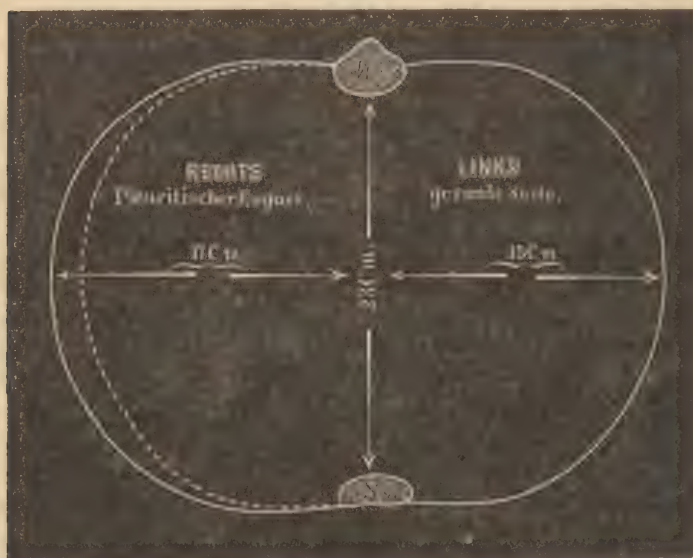


But this position of the body is by no means constant. It will be noticed so much more certainly the greater the amount of fluid; it may be prevented by the great tenderness of the affected side. In such cases, the patients sometimes assume a diagonal position upon the affected side, midway between complete dorsal and lateral decubitus. Complete dorsal decubitus when the exudation is slight, and decubitus upon the healthy side when absorption begins, are also observed.

The affected side usually presents a distinct increase in the circumference of the thorax, its degree depending on the amount of the exudation and the yielding character of the thoracic walls. The ectasia is sometimes confined to the lower part of the pleural space, but if the exudation is abundant, the entire thorax on the affected side takes part in the dilatation.

The intercostal spaces appear broader but less distinct than on the healthy side, and are sometimes almost entirely effaced. In rare cases,

FIG. 90.



Cyrtometer curve in extensive pleurisy of the right side in a man, æt. 40 years (at the level of the nipples). *St*, sternum, *W*, seventh dorsal vertebra. The dotted line denotes the difference in the size of the right and left sides of the thorax.

the intercostal spaces bulge outwards. The skin is more shining and is sometimes thrown less readily into folds than on the healthy side. The shoulder and the acromial end of the clavicle are unusually high upon the affected side and the spinal column is not infrequently slightly scoliotic, with the convexity towards the side of disease.

The degree of distention is readily determined with a tape measure, but it should be remembered that the circumference of the right side of the thorax is normally one to two cm. greater than that of the left side. It is rare to find an increase of more than five cm. as the result of pleurisy. A good representation of the distention may be secured by means of a lead or copper wire, or Woillez' erytometer (Fig. 90). Verliac found

that in children the healthy side was larger than the affected side, on account of vicarious emphysema.

The affected side of the chest takes very little or no part in the respiratory movements; in the former event, they often begin later than on the healthy side, and may be irregular and interrupted.

Inspiratory retraction of the epigastrium is sometimes observed, when the weight of the inflammatory fluid presses the diaphragm downwards, so that it projects with a convex surface into the abdominal cavity. If inspiratory contraction of the diaphragm occurs under such circumstances, the points of origin of the diaphragm must necessarily be drawn inwards.

The respirations are almost always considerably increased in frequency. This is usually the result of various factors. In the first place, the compression of the lung diminishes the respiratory surface, so that the patient attempts to secure compensation by increasing the number of respirations. Furthermore, if pain is felt, the respirations are more superficial and therefore more hurried. In addition, the respirations are hurried when the heart is pushed strongly to one side, for not alone is the mobility of the healthy lung impeded, but circulatory disturbances must also be produced. It should also be taken into consideration that the diaphragm is sometimes pushed strongly downwards, or that the intercostal muscles are slightly paretic, as the result of serous infiltration. Febrile movement is also a cause of increase in the number of respirations.

Very striking phenomena are produced when the exudation gives rise to displacement of adjacent organs. This is influenced, not alone by the amount of fluid exudation, but also by the mobility of the organs. An organ will not undergo displacement if it is fixed by adhesions.

The earliest signs of displacement are usually presented by the heart. These are particularly striking in left-sided pleurisy when the cardiac movements are visible to the right of the sternum. The displacement is sometimes so marked that the cardiac movements are seen on the outside of the right mammary line. As a rule, the heart *in toto* is pushed to the right, while torsion around its long axis is exceptional. Hence it is not the apex, but the right border of the heart which is observed pulsating on the extreme right. If the heart is pushed to the left by exudation into the right pleural cavity, the apex beat passes beyond the left mammary line, and may even be visible in the axillary line. As a rule, it is also lower than normal on account of the depression of the diaphragm.

Displacement of the liver may be looked for particularly in right-sided pleurisy. The lower border of the liver appears occasionally as a slight prominence, which is unusually low and presents respiratory locomotion.

Pulsation of the affected side of the thorax occurs in rare cases, but this has been observed hitherto only in empyema of the left side. According to Comby, it occurs only when the lung has become non-aërated from compression, and is at the same time adherent to the pericardium.

Palpation furnishes important data in diagnosis, particularly with regard to vocal fremitus. This is either diminished or abolished wherever fluid is situated beneath the walls of the thorax.

Very thin layers of fluid are capable of changing the vocal fremitus, but we must remember that it is normally greater on the right side than

on the left. If the ulnar border of the hand is alone employed, cautious palpation will enable us to determine the upper border of the exudation by the diminution of fremitus. The vocal fremitus is so much weaker the thicker the layer of fluid; hence it is sometimes absent over the lower portion of the chest.

If there are adhesions within the fluid, vocal fremitus is retained at the insertion of the adhesions into the costal pleura. Lépine states that he has even observed increase of vocal fremitus. Under such circumstances the fibrous bands evidently convey the waves of sound to the walls of the thorax.

In palpation of the thorax, attention should also be paid to the feeling of increased resistance of the thorax. This is tested most simply by immediate percussion of the thorax with the flexed index or middle finger. A greater resistance is felt by the finger wherever fluid is in contact with the chest-walls, and we are thus able to determine the height of the exudation with tolerable exactness.

Pressure upon the chest sometimes leaves a shallow depression, the result of œdema of the affected side of the chest. It was formerly supposed that this is indicative of a purulent exudation, but it is also noticed when the exudation is serous. If the exudation is very extensive and causes compression of the vena azygos or vena hemiazygos, a unilateral stasis œdema may be produced. The œdema first referred to must be regarded as an inflammatory œdema, *i. e.*, the inflammation of the costal pleura extends in part to the entire thickness of the chest-wall; at the periphery, *i. e.*, the skin, the exudation contains only fluid constituents. Stasis œdema is usually more extensive than inflammatory œdema, and may extend to one side of the abdominal parietes.

Fluctuation is observed in very rare cases; it is most apt to occur when the intercostal spaces bulge outwardly. In order to detect fluctuation, the palpating fingers should be applied very close to one another. Verlias found that, in children, when the fingers were pressed into an intercostal space, and at the same time the hypochondrium compressed from below, the impact of the fluid against the thorax was perceptible above.

In determining the area of pain, palpation is employed in the same manner as in dry pleurisy.

Palpation serves to confirm certain phenomena which are visible to the eye; for example, the slight part taken by the chest-wall in the respiratory movements. Digital examination is also extremely important in demonstrating displacement of the heart, liver, and especially the spleen. In the latter event, an elongated rounded body, usually of soft consistence, appears in the left hypochondrium. If the diaphragm is pushed downwards to a marked extent by the weight of the exudation, its costal origin may sometimes be felt as a convex prominence along the hypochondrium. This prominence sometimes appears suddenly during a rapid movement of the body, or in coughing. If this occurs on the left side, we must be careful to avoid mistaking the diaphragm for the spleen. If the diaphragm protrudes on the right side, a furrow may form between the diaphragm and liver; this may not alone be felt, but is also occasionally visible. This phenomenon may be important in the differential diagnosis between enlargement of hepatic dulness and a pleuritic exudation situated above the liver.

The symptoms on percussion, associated with the character of the vocal fremitus are the decisive features in the diagnosis of fluid pleurisy. Without the demonstration of dulness, the diagnosis is impossible. If



there is only a slight accumulation of fluid, dulness must first be sought over the posterior and inferior portions of the chest. The more the exudation increases in amount the higher the dulness rises along the spinal column, but at the same time it extends laterally, and finally anteriorly. When the exudation was very abundant, I have often found, immediately adjacent to the spine, an area of resonance, forming a vertical strip about three cm. in width.

The upper limit of dulness is usually higher, near the spinal column, than it is anteriorly, when the patient is in a recumbent position. But this is by no means constant, and I have recently observed several cases of pleurisy with a moderate amount of effusion, in which the limit of dulness was considerably higher anteriorly than posteriorly.

Damoiseau noticed that the upper limit of dulness does not always

Fig. 91.



Boundary of dulness in moderate pleuritic exudation of the left side in a man *æt.* 21 years.

run in a straight line, but presents curved elevations on the sides. Gerhardt attributed this phenomenon to the zigzag origin of the muscles, and the consequent unequal thickness of the chest-walls. Leichtenstern observed the phenomenon only in pleurisy which was undergoing absorption, and explains it on the theory that the lungs do not dilate uniformly in all directions. In opposition to this opinion, it may be stated that we have observed the symptom in question in recent pleurisy. It seems most probable to us that it is the result of irregular pleuritic adhesions.

The beginning of dulness and the height of the exudation do not coincide exactly with each other. The dulness usually begins 1.5–2.0 cm. higher than the fluid, probably because those parts of the lung

which are immediately above the fluid are compressed and partly deprived of air. If the exudation is not too extensive, respiratory movements of its upper border may be noticed. Changes in the level of dulness on changing the position of the body are much rarer, on account of the adhesions which are usually present along the upper border of the fluid. At all events, we must always wait some time before these changes in dulness appear.

When the exudation is moderately large in amount, the percussion sound is sometimes deep and tympanitic over the anterior surface of the thorax in the first and second intercostal spaces. This is explained on the ground that the lungs, in consequence of retraction and compression, are in a condition of abnormally low tension.

If the exudation is so extensive that dulness is found over the entire side of the chest, the conditions for the development of Williams' tracheal sound are furnished. This is found in the first and second intercostal spaces, and more frequently on the left than on the right side. In addition to the dulness we here find a tympanitic sound, whose pitch changes on opening and closing the mouth. Wintrich noticed that the tympanitic sound occasionally had a metallic quality. Under the circumstances referred to, the vibrations produced by percussion are conveyed through the lung, which has been compressed by the exudation, to the air contained in the main bronchus and hence give rise to the tympanitic sound which is produced in all smooth-walled cavities.

The cracked-pot resonance is also heard occasionally in this locality if the percussion beats are vigorous and short. The air is then forced with abnormal rapidity through the narrow rima glottidis, so that a hissing murmur of stenosis is produced. This is more distinctly audible if the mouth is kept wide open, and the sound may then appear to start from the buccal cavity.

Cracked-pot resonance is also heard not infrequently in fluid pleurisy under other conditions. It is heard quite often when the exudation is small in amount, either immediately above or below the limit of dulness.

Fraentzel and Traube have called attention to an important sign in the diagnosis of left-sided pleurisy, viz., diminution or disappearance of the semilunar space.

The semilunar space is a zone which presents a tympanitic percussion sound, and is situated beneath the area of cardiac dulness. It begins internally at the fifth or sixth left costal cartilage, and its lower border extends along the left lower border of the thorax to the ninth or tenth rib. Its upper border is in contact with the lower border of cardiac dulness and forms a curved line, with the convexity directed upwards. This space corresponds anatomically to the fundus of the stomach. If the diaphragm (and with it the stomach) is depressed by the weight of the exudation, the semilunar space must diminish in size, and it may even disappear.

More or less feeble vesicular breathing is heard in many cases over the exudation. The respiratory murmur is diminished, not alone on account of the diminution of the respiratory movements on the affected side, but also because the fluid exudation is situated between the lung and the chest-wall. The respiratory murmur may also disappear entirely.

Bronchial breathing may be heard over the exudation when the air has been forced out of the lungs as the result of compression. The bronchial breathing may be extremely loud, but if the exudation is very

thick, it is enfeebled. It generally diminishes in intensity at first during inspiration, later during expiration.

A metallic respiratory murmur has been repeatedly heard in uncomplicated fluid pleurisy.

Râles may be entirely absent in pleurisy. Their development usually depends on the co-existence of bronchitis, and they vary according to the character of the latter.

The voice sounds are diminished in intensity if a considerable accumulation of fluid is present in the pleural cavity. If pleuritic adhesions are present, the voice sounds may be intensified in those localities in which the adhesions are inserted into the costal pleura. The diminution of bronchophony affects not only the intensity but also the distinctness of articulation of the voice.

The bronchophony may be intensified if the exudation is slight in amount. This is owing to the fact that the lungs, being compressed by the exudation, are a better conductor of sound, and at the same time the amount of fluid is too small to diminish the sound. Increased bronchophony is sometimes noticed when the exudation is four cm. in thickness. It is observed not infrequently above the fluid.

The voice sounds, on auscultation, sometimes have a nasal bleating character (*ægophony*).

*ægophony* may also be heard over cavities and infiltrations of the lungs. It is more frequent in moderate than in very large exudations. Sometimes it is found at the level of the exudation in a zone which extends like a girdle around the thorax, from the spine to the region of the nipple; sometimes it is found only over a circumscribed region, usually in the axillary space. It is explained on the ground that the pressure of the fluid on the lungs readily compresses the peripheral bronchi, and that the waves of sound succeed temporarily in passing the site of compression and reaching the surface of the chest. In large exudations the compression of the bronchi is generally too great to be overcome by the waves of sound, and *ægophony* is not produced under such circumstances. It often makes its appearance when an extensive effusion begins to undergo absorption, and, on the other hand, it is lost when an exudation of moderate amount becomes larger.

Bacelli attaches great importance to auscultation of the whispered voice. It is said that in fluid pleurisy the whispered voice is heard upon the affected side, while it is absent upon both sides in other pleural effusions. This does not accord with my own experience. I have not found the whispered voice in all serous exudations, and, on the other hand, I have observed it in some cases of purulent and hemorrhagic exudations. I have also noticed it occasionally on the healthy side, and it is also found over cavities and pulmonary infiltrations.

A pleuritic friction sound is heard not infrequently in fluid pleurisy. It is heard at the beginning of the disease while it is still a dry pleurisy, and also along the upper border of the fluid. It sometimes denotes beginning absorption of the fluid; in other cases it is evidence of extension of the inflammatory process. Its interpretation in one or the other sense depends upon other symptoms.

The cough may depend upon co-existing bronchitis or upon inflammatory irritation of the pleura. The latter cause is especially active at the beginning of the inflammation. As a rule, the cough is so much more



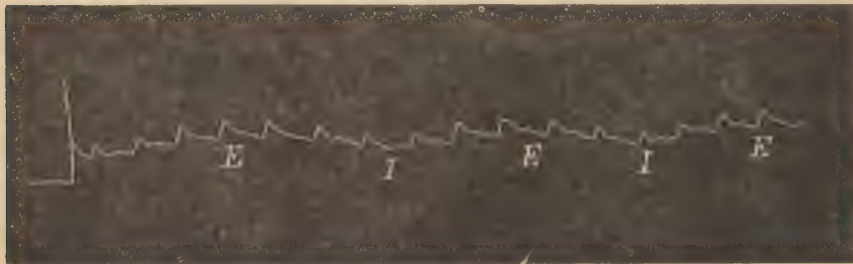
violent the more acute the character of the disease. If the pleurisy develops slowly, cough may be almost entirely absent.

The general symptoms in pleurisy with effusion are very numerous.

Not infrequently we notice changes in the color of the skin. If the fever is considerable and the exudation not extensive, the face assumes the red color of fever. If the amount of fluid is large, the color of the face becomes cyanotic. If the exudation is purulent, the patients often have an extremely pale, almost cachectic appearance, which is sometimes present from the beginning, sometimes develops slowly. If the pallor develops suddenly while the signs of a fluid exudation are developing in an acute manner, it may be assumed with some degree of certainty that the fluid is bloody. Indeed, the symptoms of a severe internal hemorrhage may appear (frequent and small pulse, cool skin, low bodily temperature, tinnitus aurium, dizziness, nausea, syncope).

The nutrition of the body depends upon the causes and duration of the disease. In chronic pleurisy the emaciation may be so marked that the patients look like those suffering from phthisis. This suspicion

FIG. 92.



Pulse curve of the right radial artery in left-sided, serous pleurisy, in a man aet. 41 years. The exudation extended to the spine of the scapula. I, inspiration. E, expiration.

may be strengthened by the occurrence of profuse night sweats, particularly in empyema.

At the same time, the bodily strength diminishes. Anorexia, increasing emaciation, and feebleness are not infrequently the sole symptoms, but an objective examination reveals a usually purulent exudation as the cause.

Elevation of the bodily temperature may be entirely absent during the course of fluid pleurisy. Even in empyema I have observed an apyrexial course for weeks. As a rule, the disease begins with one or more chills or repeated chilly sensations. This may be followed by a continued, subcontinued, or remittent fever. No positive conclusion concerning the character of the fluid may be drawn from the fever curve, though it is true that purulent exudations exhibit a greater tendency to remittent and even hectic fevers than serous exudations. Without any change in the other symptoms, we not infrequently find sudden and unexpected rises of temperature in the midst of an apyrexial course of the disease. The fever sometimes terminates suddenly, sometimes gradually.

The pulse is almost always increased in frequency. If fever is present, the rapidity of the pulse is usually greater than that which would correspond to the increased temperature. Hence the pleurisy *per se* accelerates the frequency of the pulse. This is owing to the elevation of blood

pressure and the obstruction which the exudation, by compressing the lungs, necessarily presents to the pulmonary artery. The pulse is usually small.

In two cases Leichtenstern observed *pulsus paradoxus*, the radial pulse almost disappearing at each deep inspiration. As a rule, the respirations have an unusually active influence upon the volume of the pulse in exudative pleurisy (Fig. 92). The height of the pulse-wave diminishes at each inspiration. In a case of right-sided empyema and aortic aneurism, Douglas Powel found that the pulse was absent in the right radial and carotid arteries, and did not appear until the fluid was removed by operation.

General œdema is rarely observed. It may be looked for if the strength has suffered from long-standing pleurisy, or if protracted suppuration has given rise to amyloid degeneration of the large abdominal glands, or, finally, if the primary disease, especially malignant neoplasms, favors the development of œdema.

Symptoms of circulatory disturbance in the cranial cavity are sometimes observed, the patients complaining of a rush of blood to the head, ringing in the ears, and a feeling of dizziness. If the fever is very high, cerebral symptoms similar to those of other febrile diseases may appear, and in children the fever is sometimes the cause of general convulsions.

Wille reports two cases of insanity which developed during an attack of pleurisy.

The patients complain not infrequently of a peculiar pressure and even pain in the region of the stomach, evidently the result of the pressure of the exudation on the diaphragm and stomach. I have repeatedly observed vomiting, which must also be attributed to mechanical irritation of the stomach.

The liver is not infrequently found depressed, and at the same time more resistant and enlarged.

The amount of urine is almost always considerably diminished, because the tension in the aortic system is diminished on account of the circulatory disturbances produced by the exudation. The urine is high-colored, has a high specific gravity, and a reddish sediment is often noticed; it may contain albumin and casts as the result of stasis in the renal veins.

Maixner found peptone in the urine of empyema, and Brieger found large amounts of phenol in the urine of ichorous pleurisy.

The complications of pleurisy may be the result of a peculiar localization of the inflammation, but depend especially upon the amount and character of the exudation.

With reference to the site of the inflammation we must refer particularly to diaphragmatic pleurisy, in which the inflammation is confined mainly to that portion of the pleura which lines the diaphragm.

The disease generally begins suddenly with severe subjective symptoms. The patients complain particularly of pain in the hypochondrium, extending to the back and even to the region of the shoulder. A forced position of the body is sometimes noticed, viz.: dorsal decubitus, lateral decubitus on the healthy side, or abdominal decubitus as was noticed by Ferber. Many patients complain of pain in swallowing, as

soon as the food passes the œsophageal foramen. If the inflammation is located on the left side, vomiting, singultus, and gastric pain are often noticed, while jaundice has been repeatedly observed in right-sided pleurisy, probably because the respiratory movements of the liver, and hence the discharge of bile, are interfered with. The lower border of the thorax, particularly the last intercostal space near the spinal column, is usually extremely sensitive on pressure. It is also said that the outer side of the sternomastoids, corresponding to the cervical portion of the phrenic nerve, is very painful on pressure. The lower part of the thorax on the affected side is almost immovable during respiration, and the respiratory murmur there is feeble. The dyspnoea is usually very considerable, while fever is often absent. Pleuritic friction sounds are sometimes heard over the lower border of the lung, on the left side, for example, over the greater part of the semilunar space. If no exudation is present, the diaphragm is usually very high. Abnormal phenomena on percussion are absent in many cases because a not inconsiderable amount of exudation may collect between the diaphragm and base of the lung without coming in contact with the chest-walls.

Left-sided pleurisy is complicated, with relative frequency, with pericarditis. Endocarditis also develops occasionally; in one case Wilks describes ulcerative endocarditis after empyema.

Sudden syncope and death have been noticed a number of times in pleurisy with effusion. This accident either occurred spontaneously or after bodily exertion, for example, rapid sitting up in bed, violent coughing, straining at stool, etc.

This accident depends on various causes. In many cases there is a sudden anæmia of the brain and heart. In others, we have to deal with embolic processes, usually secondary to cardiac thrombosis, the development of which is favored by the slow circulation. According to the situation of the thrombus, the embolus may enter the pulmonary artery and its branches, or be conveyed to the brain. Occlusion of branches of the pulmonary artery is especially dangerous in pleurisy, because the lung upon the affected side has been excluded to a greater or less degree from respiration. Bartels attempted to show that sudden deaths in pleurisy are the result of flexion of the inferior vena cava, particularly in left-sided pleurisy. But Leichtenstern proved that the frequency of this lesion as a cause of sudden death has been greatly overestimated. Finally, the sudden death may be the result of degeneration of the heart muscle. This is especially apt to occur when the fever is high, or changes are present which point to severe infection, while at the same time the amount of exudation entails increased labor upon the heart.

If the amount of exudation is excessive, there is danger of suffocation. Not alone is the lung upon the affected side deprived of air, but the other lung is also affected. In addition, the heart may be displaced to such an extent that death is inevitable. Pulmonary œdema sometimes develops towards the close of life.

Perforation of the pus often takes place in empyema if it is not allowed to escape by operative means. The perforation may occur externally, into the lungs, or other viscera.

As a rule, we can easily foretell the perforation of the pus externally. At first a circumscribed œdema of the integument of the chest is noticed; then this part becomes more prominent, and a sensation of fluctuation is felt on palpation. During inspiration, the swelling becomes smaller; during expiration and coughing, it grows larger. The tumor may gen-



erally be diminished in size upon pressure, but this must be done very carefully in order to avoid perforation of the skin. Pulsation of the tumor in all directions is sometimes observed.

When perforation of the pus through the skin is impending, the overlying skin grows red, becomes thinner and thinner, and either ruptures suddenly in a broad rent through which the pus flows in a large stream, or the solution of continuity is gradual, and the pus trickles slowly. A thoracic fistula has thus been produced. The daily amount of pus discharged may be more than one litre, especially at the beginning. The pus is usually laudable, but after a time it often acquires a sour odor like that of buttermilk.

Some authors state that the perforation most frequently occurs anteriorly in the space between the sternal end of the lower costal cartilages and the edge of the sternum, because the external intercostal muscles are absent in that locality. My own experience does not accord with this statement. Almost all the spontaneous thoracic fistulæ under my observation were situated in the fifth or sixth intercostal spaces between the mammary and axillary lines.

The pus occasionally follows the skin for a long distance, and makes its appearance remote from the thorax. I have recently seen a thoracic fistula situated directly above the posterior portion of the crest of the right ilium. In some cases the fistula has been observed above Poupart's ligament, and even in the popliteal space.

The external and internal fistulous openings are rarely situated opposite to one another, even when the pus follows the straightest possible path. The internal opening is usually much larger than the external.

For a few days the perforation is often apparently followed by favorable subjective and objective changes. The patients feel easier, breathe more freely, and the fever sometimes ceases.

It is rare that the pus is gradually discharged *in toto*, the fistula closes, and recovery occurs. As the rule, the pus again accumulates, the patients gradually lose strength, and finally die with hectic symptoms, perhaps not until after the lapse of years. Long continued suppuration may also give rise to waxy degeneration of the large abdominal glands and intestines, so that the patients succumb after dropsical symptoms, or obstinate and profuse diarrhœa. The fistula not infrequently closes temporarily. The patients feel tolerably well for weeks, and even months, then anorexia, chills, sweats, fever are observed, the fistula re-opens, and the daily discharge of pus again begins. The bones of the thorax are occasionally implicated, and the ribs undergo caries and necrosis.

Perforation of an empyema into the lungs may take place suddenly or gradually; it is always preceded by erosion and destruction of the pulmonary pleura. If the perforation takes place suddenly, the patient begins to expectorate large amounts of pus as soon as the pus which has entered the lung reaches the lumen of a larger bronchus. The large bronchi not infrequently are flooded so quickly and completely that there is great danger of suffocation, and the patient may even die before pus is expectorated. This is particularly apt to occur if the perforation occurs during sleep, or if the pus runs back into the bronchus of the intact lung.

The amount of pus expectorated may be very considerable, and may far exceed one litre. Friedreich and Biermer found that it contained cholestearin tablets and hæmatoidin crystals; in one case I found the

Charcot-Neumann crystals. The expectorated fluid consists in the main of pus-corpuscles, which are partly fatty.

As a rule, the expectoration continues for several days. At first it has a sour odor, but later it often has a stinking, putrid odor. This is especially apt to occur when the flow into the bronchi diminishes, and the masses of pus stagnate and decompose in the tubes. At all events, we may not infer from the putrid odor of the purulent sputum that the exudation itself has undergone decomposition. This is not apt to occur unless air has entered the pleural cavity through the opening of perforation, and has given rise to pneumothorax. Such an event occurs very rarely, because the fistula generally has a valve-like conformation so that it is only passable from the pleura to the lungs.

The expectoration of pus sometimes stops temporarily, but high fever then occurs, the exudation in the pleural cavity increases, and after a time another perforation takes place. This series of events may be repeated a number of times.

The perforation occurs more frequently through the middle and upper than through the lower lobes of the lungs.

Perforation of an empyema into the lungs may also take place in a more gradual manner. If the pulmonary pleura is destroyed in one or more places, the pus slowly permeates the lung as if it were a sponge, and then passes into the bronchi. Under such circumstances a grayish-yellow or greenish-yellow sputum is expectorated in large quantities (sometimes more than one hundred ccm.). This contains a large proportion of pus, and on standing deposits a granular layer of sediment composed almost entirely of pus-corpuscles.

Various complications of pleurisy appear when the pus perforates into the pericardium, mediastinum, a main bronchus or the trachea, the other pleural cavity, œsophagus, stomach, or intestines. These complications are recognized in part by the secondary inflammations, in part by the appearance of large quantities of pus which are discharged by vomiting, coughing, evacuation from the bowels, or by the urine if the rupture takes place into the urinary passages.

Acute nephritis is observed not infrequently as a complication.

The duration of exudative pleurisy depends mainly on the amount and character of the exudation. Moderate exudations, *i. e.*, those reaching to the middle of the scapula, are generally absorbed in three to six weeks. Purulent exudations are rarely absorbed spontaneously, and the duration of the disease depends upon the operative interference. Remissions and exacerbations are not infrequent, both as regards the symptoms and the objective changes in the thorax. Too early getting up is often followed by a marked exacerbation. Exudations which have been present for a long time are sometimes absorbed with striking rapidity when the patients undergo great losses of water; for example, in profuse diarrhœa or sweating.

Traube described pleuritis acutissima as a special form of pleurisy. It runs the course of an infectious disease with severe general symptoms: high fever, clouded sensorium, dry, fissured tongue, meteorism, enlargement of the spleen, diarrhœa, and roseola. It may rouse the suspicion of typhoid fever.

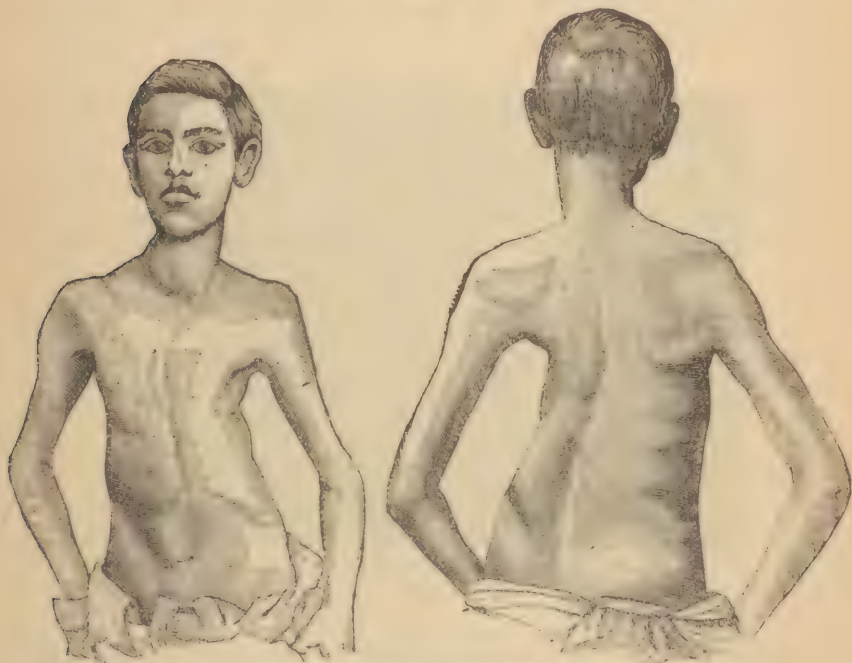
The terminations and sequelæ of exudative pleurisy are very numerous.

As a matter of course, the most favorable termination is the com-

plete, spontaneous absorption of the exudation, and a return to the *status quo ante*. This occurs only in serous pleurisy. The more profuse the serous exudation, and the longer it has lasted, the less can complete restoration be looked for.

Pleuritic adhesions are very often left over, and may give rise to subjective and objective changes for years, or even for life. During deep respirations, or on bodily exertion, stitches are felt in the chest, and the patients may also complain of shortness of breath. The dyspnoea will be so much more marked the more extensive the adhesions and the more the respiratory mobility of the lungs is impaired. Special consideration should be paid to obliteration of the complementary pleural spaces, whose recognition was discussed on page 343. Total synechia implicates the

FIG. 93.



Retraction of the left thorax after pleurisy in a boy æt. 18 years. After Ried.

heart, and sometimes, after the lapse of years, gives rise to a fatal termination with symptoms of heart failure. Adhesions and pleural thickenings of a certain extent make themselves noticeable by dulness, diminished vocal fremitus, and respiratory murmur.

If fluid pleurisy has lasted a long time, and has compressed the lung, adhesion and obliteration of the compressed alveoli develop not infrequently, and may always prevent to a greater or less extent the distention of the lungs. If complete absorption of the fluid now occurs, the thoracic wall must be driven inwards to an extent equal to the loss of distensibility experienced by the lung.

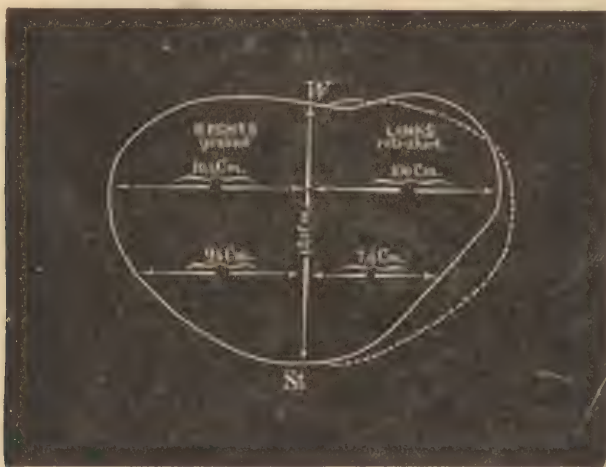
The affected side of the thorax appears sunken, even to the naked eye (Fig. 93). This is especially manifest in the lateral region, at the level



of the sixth and eighth ribs. The intercostal spaces are narrower than on the healthy side, and, if the retraction is very marked, the ribs may come in contact with one another. The inner surface of an upper rib, particularly in the lower part of the thorax, may project over a portion of the outer surface of the rib lying directly below. The nipple is usually lower than on the healthy side. The shoulder is also much lower, and the spine is scoliotic, with the convexity directed towards the healthy side. As a general thing, the scapula is rotated on its long axis, so that the inner border appears lifted up from the chest-wall. The affected side does not take much part in the respiratory movements.

The resistance of the thorax is almost always increased; the vocal fremitus is diminished. The circumference of the affected side is diminished, the difference between the two sides being usually two or three cm.; in extreme cases the difference may amount to nine cm. It must be remembered, however, that the right side is normally from one to two cm. larger than the left.

FIG. 94.



Cyrtometer curve in retraction of the left thorax after pleurisy in a girl at. 12 years. The dotted line shows the difference in the circumference of the thorax on the two sides. W, spinal column. St, sternum (at the level of the fourth costal cartilage). One-quarter natural size.

There is usually dulness on percussion over the retracted side. Auscultation shows diminished vesicular breathing, or bronchial breathing may be heard if a considerable portion is entirely empty of air.

Retraction of the thorax is often the result of several factors. The greatest effect is produced by the external atmospheric pressure. Retraction of pleuritic thickenings also plays a certain part, as is shown by the fact that thoracic retraction sometimes continues for a long time after absorption of the fluid exudation is complete. The pleuritic callosities sometimes attain a thickness of more than three cm., and have a cartilaginous consistence; they may be calcified in places, or produce deep furrows upon the surface of the lung (pleuritis deformans). In one of my cases calcification of the new membranes had advanced to such an extent that the left lung was completely enveloped in a calcareous capsule.

If the distensibility of the lungs is very much impeded, the retrac-

tion of the thorax is associated not infrequently with displacement of adjacent organs. In retraction of the left side the heart moves into the left thorax, so that its apex may reach the axillary line. It may also be situated unusually high, so that the semilunar space is necessarily increased in size. In retraction of the right side the upper border of the liver is found to be unusually high. The greater the obstruction to the dilatation of the lungs, and the more unyielding the thorax, the more marked will be the displacement of the organs. It is absent or very slight when the organs which have been pushed in the opposite direction by the exudation are fixed by adhesions. This is most marked with regard to the heart, especially in pleurisy of the left side. Under such circumstances we may find retraction of the left thorax, while the heart pulsates in the right mammary line. This abnormal position usually persists during the remainder of life.

If adhesions have formed in the upper parts of the lung, they may surround and paralyze the recurrent laryngeal nerves, usually upon the right side. In carcinomatous pleurisy, paralysis of this nerve may be the result of compression by carcinomatous lymphatic glands. Many of the patients succumb finally to pulmonary phthisis. Tubercular changes sometimes follow the pleurisy very rapidly, sometimes months, or even years elapse. Serous pleurisy is more to be dreaded in this respect than the purulent form, especially if the latter has been operated upon at a sufficiently early period. But if we wait too long, the fluid constituents of the pus may be absorbed while the cellular elements become dry and cheesy, in part are calcified, become infected with bacilli, and finally give rise to an outbreak of diffuse tuberculosis. The danger of miliary tuberculosis after serous pleurisy is said to be especially great if the fluid is absorbed very rapidly. Those individuals who have a phthisical family history are particularly endangered. It should also be mentioned that it is not always the lung whose pleura was inflamed which becomes tuberculous.

Empyema is sometimes associated with metastatic abscess of the brain. If empyema necessitatis has developed and suppuration is protracted, waxy degeneration sometimes occurs in the spleen, liver, and kidneys, and is shown by enlargement of these organs, albuminuria, and cutaneous œdema. Chronic Bright's disease (generally chronic parenchymatous nephritis) sometimes develops; paranephritis has also been observed as a complication and sequel of pleurisy.

IV. DIAGNOSIS.—Dry pleurisy is easily recognized if a pleuritic friction murmur is present, since its occurrence without pleurisy has not been positively demonstrated. The friction sound is not very apt to be mistaken for similar sounds.

Sonorous bronchial râles are the most apt to lead to mistakes. If pressure with the stethoscope increases the intensity of the sounds they are pleural in character, since the sonorous râles remain unaffected by this manipulation. Sonorous râles usually disappear or undergo considerable change after vigorous coughing, while the friction sound persists. As a rule, the râles present a more continuous character and are more widespread than the pleuritic friction sound.

If a dry pleurisy is confined to the vicinity of the pericardium, it may be mistaken for pericarditis, because the friction sounds are not alone dependent on the respiratory movements, but also on the cardiac movements (pleuro-pericardial or external pericardial friction sound). The following are the distinguishing features:

*a.* The respiratory portion of the pleuro-pericardial friction sound predominates over the cardiac portion, so that it may be intensified or diminished by deep or superficial respirations.

*b.* If the patient holds his breath, the pleuro-pericardial friction sound usually ceases very soon. It grows gradually feebler and ceases after three to six cardiac contractions. It reappears when respiration is resumed.

*c.* The murmur is abolished so long as the patient holds his breath after taking a full inspiration; the pericarditic friction sound is intensified under such circumstances.

*d.* The pleuro-pericardial sound is heard generally near the apex (corresponding to the lingual process of the lung), at all events it is especially frequent at the left border of the heart, while endo-pericarditic murmurs are usually more diffused, and first appear over the conus of the pulmonary artery.

If the pleuritic friction sound is wanting, the diagnosis of dry pleurisy is much more difficult, inasmuch as it is based solely upon the pleuritic pain. It is readily mistaken for intercostal neuralgia, inflammation of the ribs, muscular rheumatism, and dermatitis.

In intercostal neuralgia the pain is generally intermittent, often in periodic attacks; it is generally confined to a single intercostal space, and extends often from the spine to the sternum. Valleix's pressure points should also be taken into consideration in intercostal neuralgia. There are usually three. One alongside the spine, where the nerve emerges from the spinal canal, a lateral point in the middle of the intercostal space, and a sternal point immediately adjacent to the sternum.

In the differential diagnosis between dry pleurisy and caries of the ribs, it should be remembered that the pain in the latter affection is confined to a single rib, that it becomes very intense on pressure upon the rib, while the pleuritic pain is increased by pressure in the intercostal space. As a rule, the affected bone is slightly swollen and the skin above is reddened.

The differentiation between dry pleurisy and rheumatism of the thoracic muscles may be very difficult. If the latter disease is very extensive, the patients are sensitive to pressure on all parts of the chest, and since pain on respiration may also be present, it may be associated with a feeling of want of breath or even dyspnoea. If the affection is confined to the large muscles, the pain is increased when these muscles are compressed between the fingers.

Inflammation of the skin is recognized by the cardinal signs of inflammation, viz., redness, swelling, and heat.

In the diagnosis of fluid pleurisy, we must determine not alone the presence of an inflammatory fluid in the pleural cavity, but also the character of the fluid.

The differentiation of pleurisy from infiltration of the pulmonary alveoli with solid masses is usually very easy. The condition of vocal fremitus alone is very often sufficient. In pleurisy it is diminished, in pneumonia it is increased. In the latter disease, vocal fremitus is diminished only when fluid pleurisy is also present or when the bronchus leading to the pneumonic spot is occluded by secretion. In the latter event, however, the secretion will usually be removed by a vigorous spell of coughing, and vocal fremitus will again be increased.

In doubtful cases we must pay attention to the character and shape



of the area of dulness. In pleurisy the dulness usually increases from above downwards; if the dulness is more intense superiorly, it would favor the diagnosis of pneumonia. As a rule, the area of pleuritic dulness diminishes from behind and above to the front and below, while the upper border of pneumonic dulness may run an entirely irregular course. In left-sided pleurisy the condition of the semilunar space is important; in pleurisy it grows smaller or disappears, in pneumonia its dimensions are not appreciably affected. It is only when the pneumonic infiltration is very extensive that the size of the lung is increased to such an extent as to cause a diminution—usually slight—of the semilunar space, and even displacement of the heart. In some cases, furthermore, the relation of the intensity of the bronchial breathing to the intensity of the dulness is important in differential diagnosis. Very loud bronchial breathing, together with very marked dulness, is evidence against the existence of an extensive pleuritic exudation. The rusty sputum is decisive in the diagnosis of fibrinous pneumonia. Considerable displacement of adjacent organs and dilatation of the thorax are foreign to pneumonia.

Insurmountable obstacles may be presented by the differential diagnosis of extensive pulmonary tumors and pleurisy. If the tumors occlude the bronchi, we will find extensive dulness and loss of vocal fremitus. The following data are useful in differential diagnosis: absence of displacement of adjacent organs and of dilatation of the thorax. The differentiation between pleurisy and extensive tumors of the pleura may also be impossible.

Circumscribed pleurisy may give rise to diagnostic mistakes if it is situated near the heart or immediately above the spleen. In the former event it may be mistaken for pericarditis. As a rule, however, the boundary of the area of dulness is more irregular than in pericarditis, the changes in the apex beat characteristic of pericarditis are absent, and also the pericarditic friction murmur. If the circumscribed pleurisy is situated in the splenic region, it may be mistaken for enlargement of the spleen. One of the chief elements in the diagnosis is our ability to feel the spleen. Furthermore, an enlarged spleen presents respiratory changes of position of the area of dulness; this is absent or very slight in circumscribed pleurisy.

An accumulation of fluid in the right pleural cavity (if it is not too extensive) may be mistaken for enlargement of the liver. It is rare that enlargement of the liver occurs only in an upward direction, so that, if the lower border of the liver is situated in the normal position, a supposed hepatic dulness which begins high up should be looked upon as a pleuritic exudation. While the dulness of fluid pleurisy is generally higher posteriorly than anteriorly, it is often found in tumor, echinococcus, and abscess of the liver that the dulness is higher anteriorly and posteriorly than it is in the axillary line. Furthermore, dulness which is connected with the liver usually presents respiratory displacement, while this is absent or very slight in pleuritic dulness. In pleuritic exudation the intercostal spaces are generally effaced; in hepatic tumors they are retained, but the lower ribs are often bent outwards. Stokes called attention to the fact that in extensive pleuritic exudations a furrow often forms between the anterior surface of the liver and the lower border of the thorax, as the result of displacement of the liver. If this is very marked, the furrow cannot alone be felt, but is also visible to the eye. Frerichs has shown, however, that not alone is the fur-

row absent not infrequently (or at all events presupposes a large amount of fluid), but that it is also found in cases of tumor of the liver, if the neoplasm projects above the surface of the organ near the lower border of the thorax. The clinical history of the disease must also be taken into consideration.

If empyema necessitatis develops, superficial examination may leave us in doubt whether we have to deal with a cold abscess originating in the spinal column, with a peri-pleuritic or subcutaneous abscess, with caries of the rib or aneurism.

Disease of the spinal column may be excluded if no changes are found in that locality. In addition, other physical signs of pleurisy are found.

In peri-pleuritic abscess, *i. e.*, an accumulation of pus outside of the costal pleura, only that intercostal space over which the abscess is situated is enlarged, while the adjacent ones appear narrowed. The abscess does not increase in size in all directions on straining, coughing, and forced expiration, nor can it be diminished in size by pressure. In peri-pleuritic abscess, the boundaries of the area of dulness are entirely irregular, and air-containing tissue can occasionally be demonstrated, by strong percussion, below the abscess. If the abscess has been opened, the finger, when introduced, soon meets with an obstruction; but in empyema it is freely movable in the pleural cavity.

Subcutaneous abscesses, or abscesses following caries of the ribs are easily recognizable from their superficial position and the absence of other pleuritic symptoms.

Empyema necessitatis may be mistaken for aneurism only in cases of empyema pulsans in which the pulsations are also expansile. Kussmaul and Mueller mention the following differential points:

*a.* The aneurism is situated most frequently above and to the right side of the anterior thoracic surface; empyema pulsans is situated to the left side below.

*b.* The dimensions of the empyema change on movements of straining and respiration; this is not true of aneurism.

*c.* In aneurism the dulness is confined to the tumor; in empyema it passes beyond it.

*d.* Circulatory murmurs are usually present in aneurism, absent in empyema.

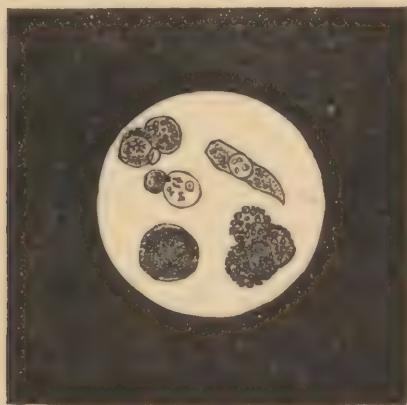
An exploratory puncture is the only certain measure for determining the nature of the pleuritic effusion. The hypodermic syringe which is employed for this purpose must be thoroughly cleaned and disinfected, and its aspirating power must be tested. If we are certain that the pleural cavity contains fluid, the needle should be inserted into an intercostal space over which dulness is found. It is well to fix the two ribs, between which we desire to introduce the needle, with the second and third fingers of the left hand, and then to push the needle in as rapidly as possible. If the introduction is made slowly the patients often twist the spinal column and thus cause movement of the ribs. In this manner there is danger of striking the rib itself with the end of the canula. In such cases no violence may be used, or the canula may break and give rise to very disagreeable complications. The canula must then be directed upwards or downwards, according to circumstances, and we will then be able to enter the desired intercostal space. The posterior surface of the thorax is best adapted for the site of puncture, because we thus

avoid the observation of the patient and may manipulate without hindrance.

If the canula is very narrow or is plugged by flakes of fibrin and the like, the puncture will be unsuccessful. The canula should then be withdrawn, cleaned, and inserted in another place. The site of puncture should be covered with adhesive plaster.

The character of the aspirated fluid (serum, pus, blood, ichor) is easily recognized. In rare cases of empyema the exploratory puncture gives rise to an error in diagnosis, because the pus-corpuscles have been deposited at the bottom, while above them is a tolerably clear serous layer. To avoid error in such cases, it has been proposed to shake the patient and thus stir up the fluid. Those physicians who care for the welfare of their patients will not carry out this humane recommendation.

FIG. 95.



Cancer cells in a pleural exudation. After Quincke.

Exploratory puncture may throw light, in some cases, upon the etiological relations of the exudation. In pleurisy following the infectious diseases, the fresh fluid has been found to contain bacteria. In the pleurisy following cancer of the pleura, desquamated cancer elements have been found in the fluid obtained by the puncture. Special attention should be paid to fatty cells, which are present in large numbers, to cells containing vacuolæ, and to cell-masses composed of fatty cells and cells containing vacuolæ (Fig. 95). The latter are apt to accumulate in the deepest layers of the exudation, so that it is well to make the puncture as low as possible.

The nature of the exudation cannot be determined by the clinical symptoms alone, because the exceptions to the rule are so frequent. The rapid formation of extensive exudations associated with signs of profound anæmia may be attributed to a bloody exudation. Hectic fever, repeated chills, sweats, and rapid exhaustion occur most frequently in empyema. Double pleurisy is usually of a purulent or hemorrhagic character.

The etiology should also be taken into consideration in making a probable diagnosis of the nature of the pleuritic exudation. Hemorrhagic exudations occur with special frequency in tuberculosis, carcinosis of the pleura, scurvy, and Bright's disease. Purulent exudations are most



frequent in pyæmic and infectious processes; the majority of cases of so-called rheumatic pleurisy are serous. Ichorous pleurisy occurs chiefly in pyæmic processes and gangrenous affections of the lungs.

Quinke and Unverricht noticed that in carcinomatous pleurisy hard cancerous nodules develop not infrequently under the skin at the site of exploratory puncture. Unverricht believes that this feature is useful in the diagnosis of pleural cancer, especially when associated with paralysis of the recurrent laryngeal nerve, the latter being the result of compression by the cancerous, enlarged tracheo-bronchial glands. Purgesz states that in carcinomatous pleurisy the intercostal spaces are sometimes narrowed as the result of degeneration of the costal pleura, and that in left-sided pleurisy the semilunar space is retained. According to this writer, hardening at the site of puncture may also occur in simple empyema.

The pleuritic effusions of childhood are most frequently of a purulent nature.

V. PROGNOSIS.—The prognosis depends mainly on the etiology and the character of the inflammation. In many cases the prognosis is unfavorable because the primary affection is not susceptible of improvement or recovery (tumors of the pleura, pyæmia, Bright's disease, chronic pulmonary diseases).

As regards the character of the inflammation dry pleurisy presents the most favorable outlook for recovery. It is only when it has occurred repeatedly and exclusively near the apex, that the prognosis should be made with great caution, because the pleural inflammation is not infrequently the result of latent phthisis.

In fluid pleurisy the prognosis depends on the character and amount of the fluid. Serous pleurisy presents the most favorable outlook. It is only in very exceptional cases that purulent pleurisy is susceptible of spontaneous absorption. But the prognosis in this form of the disease is favorable if the nature of the fluid is recognized very early and if we are imbued with the idea that recovery is only possible from the immediate discharge of the pus by means of an incision. The prognosis of ichorous and hemorrhagic exudations is unfavorable from the very nature of their causes.

The amount of fluid may give rise to danger in a mechanical way, since displacement of adjacent organs is compatible with life only when it does not pass beyond a certain grade. Moreover, very large exudations interfere with absorption, probably because the lymphatics are compressed, and their function interfered with.

A very grave prognosis should be made in those cases which run their course as pleuritis acutissima. The longer the exudation lasts and the more unchanged it is, the less favorable are the chances of absorption. If an exacerbation of the symptoms and an increase of fluid occur after a few weeks, the exudation will rarely disappear without our interference.

It is said by various authors that right-sided pleurisy is associated more frequently with pulmonary phthisis than pleurisy of the left side.

Even under the most favorable circumstances, the prognosis of fluid pleurisy should be made with a certain amount of reserve, because a fatal termination may be produced by entirely unlooked-for accidents. The prognosis as regards the future should also be made with caution, since a pleurisy which is apparently entirely cured often proves the starting-point for the development of phthisis.

VI. TREATMENT.—In a primary dry pleurisy, we should insist that

the patient stay in bed. If there are no marked general disturbances, this direction will often be opposed. It is necessary, however, in order to prevent the formation of a fluid exudation. Continued warm poultices may be applied to relieve pain in the side. The ice-bag is not tolerated so well in many cases, and relieves the pain with less certainty. If the pain is very severe, and there is no high fever, we may make a subcutaneous injection of morphine over the painful spot (℞ Morphin. hydrochlor., 1.0; glycerin. pur., aq. destil., āā 15.0. M. D. S. A half-syringeful subcutaneously). If the cough is very severe, it is better to administer the morphine by the mouth, in the form of a powder, or in combination with decoct. rad. althææ, or infus. ipecacuanhæ (℞ Morphin. hydrochlor., 0.05; sacch. alb., 0.5. M. f. p., d. t. d. No. x. S. 1 powder three or four times a day.—℞ Decoct. rad. althææ, 10 : 180; morphin. hydrochlor. 0.01; syr. simp., 20.0. M. D. S. One tablespoonful every two hours.—℞ Inf. rad. ipecac., 0.5 : 180; morphin. hydrochlor., 0.01; syr. simp., 20.0. M. D. S. One tablespoonful every two hours). Wet or dry cups, fly-blisters, irritating inunctions, for example, chloroform liniment (℞ Chloroform., 10.0; liniment. volat., 40.0. M. D. S. To be rubbed in t. i. d.) have also been recommended to relieve the pains in the chest. If there is considerable fever, or the patient is annoyed by a complicating bronchitis, the ordinary remedies may be employed.

The expectant plan of treatment should be adopted at first in a recent, uncomplicated serous pleurisy. If no change occurs in two or three weeks, absorption should be aided by the method which will be described later. If no result is obtained in four or five weeks, or the exudation is even increasing, the fluid should be removed by puncture. This may be repeated, if the fluid again accumulates; but if the fluid is purulent, different treatment must be adopted. Aspiration should also be performed if the amount of fluid is so great, at any time, that the compression and displacement of other organs constitute a source of danger.

We will now consider the individual stages of treatment somewhat more in detail.

Persons suffering from recent serous pleurisy should always keep to bed, even if the symptoms are very slight. If the cough is severe, we should give narcotics, warn the patient against sudden movements of the body, and against straining at stool. A tendency to constipation may be relieved by giving stewed fruit or mild laxatives (℞ Electuar. e senna 25.0. D. S. One to two teaspoonfuls at night.—℞ Aloes, ext. rhei, tub. jalap., āā 1.0; pulv. et succ. liq., q. s. ut fiat pil. No. 30. D. S. Three pills taken at night). The affected side of the thorax should be covered with a warm poultice. The diet should be mild and nourishing (milk, eggs, meat-soups, beer, wine). In the beginning it is unnecessary to give internal remedies, unless special indications arise.

Antiphlogistic measures have been widely employed, especially in England, to abort a recent pleurisy, but this plan of treatment is not attended with much success.

If signs of beginning absorption remain absent during the second or third week, we should administer absorbents, diuretics, drastics, diaphoretics, or tonics.

Among absorbents, the preparations of iodine occupy the first rank. If potassium iodide (10 : 200; one tablespoonful t. i. d.) is given internally, it passes into the pleuritic exudation. Tincture of iodine, potas-

sium-iodide ointment, iodoform (3 : vaselini 50) may be applied to the affected side.

Tincture of iodine should be used with care, because it produces irritation of the skin. If the epidermis scales, the iodine should not be applied again before a new, firm epidermis has formed. Preparations of iodine should also be discontinued as soon as symptoms of iodism appear (acne, conjunctivitis, coryza, burning in the throat).

Diuretics will be especially useful if diuresis is very scanty. According to circumstances, we may order digitalis combined with acetate of potash or soda ( $\mathcal{R}$  Inf. fol. digit., 1.0 : 150; liquid kalii acet., 30.0; syr. simp., 20.0. M. D. S. One tablespoonful every two hours.— $\mathcal{R}$  Inf. fol. digit., 1 : 180; kalii nitrici, 10.0; syr. simp., 20.0. M. D. S. One tablespoonful every two hours), the potash salts ( $\mathcal{R}$  Sol. kalii nitrici, 15.0 : 200; one tablespoonful every two hours.— $\mathcal{R}$  Sol. natrii nitrici, 15 : 200; one tablespoonful every two hours.— $\mathcal{R}$  Kalii tartarici, 20.0; aq. destil., 180.0; syr. cinnam., 20.0. M. D. S. One tablespoonful every two hours); or the vegetable diuretics ( $\mathcal{R}$  Acet. scillitic., 30.0; kalii carbonic., q. s. ad saturat.; aq. petroselin., 150.0; oxymel. scillitic., 20.0. M. D. S. One tablespoonful every two hours). We may also recommend drinks of carbonated waters.

As the result of increased diuresis, water is abstracted from the blood, and the latter covers its loss by absorption of the pleuritic fluid.

Cathartics are indicated only when the patients possess a certain degree of vigor, and especially when there is a tendency to constipation. A few cases have been reported in which the fluid exudation of pleurisy disappeared with remarkable rapidity when the patients were attacked with Asiatic cholera. In order that cathartics may effect this purpose, the stools should be watery in character, profuse, and repeated several times a day. The dose of the remedy may be gauged by the amount of the stools passed in twenty-four hours. Milder laxatives or stronger drastics are employed according to circumstances. We may mention the following prescriptions:  $\mathcal{R}$  Inf. sennæ comp., 180; natrii sulphuric., 20.0. M. D. S. One tablespoonful four times a day.— $\mathcal{R}$  Inf. colocynth., 1.0 : 180.0; syr. sennæ cum manna, 20.0. M. D. S. One tablespoonful t. i. d.— $\mathcal{R}$  Ol. croton., gtt. v.; ol. ricini, 30.0; gummi arabici, 7.5; ft. c. aq. destil. q. s. emulsio 150.0; syr. sennæ, 20.0. M. D. S. One tablespoonful two or three times a day.

Among the diaphoretics we must dispense, as a rule, with the use of hot water or air baths because they generally aggravate the respiratory disturbances to an unendurable degree. It is only when the exudation is very slight in amount that this plan of treatment may be adopted. For this purpose I have used a "sweat-box" arranged in the following manner. The ordinary iron-hoop arrangement to protect the bed-clothes is fastened to two side bars of wood (Fig. 96). It should extend from the feet to the region of the nipple. At the foot end and also over the hoops it is covered with oil cloth. Beneath it, at the foot of the bed, is placed a spirit lamp, firmly fastened to a wooden support and having a protecting cover of tin. The wooden support bearing the spirit lamp is fastened to the bed by means of clamps. The patient is covered with blankets and put to bed, with the spirit lamp between his legs. The lamp is lighted and the entire apparatus covered with blankets, in which the patient is wrapped up to the neck. The internal temperature of the box soon rises to 50–55° C., and the patient soon begins to perspire. The bath may be continued for an hour or even longer.

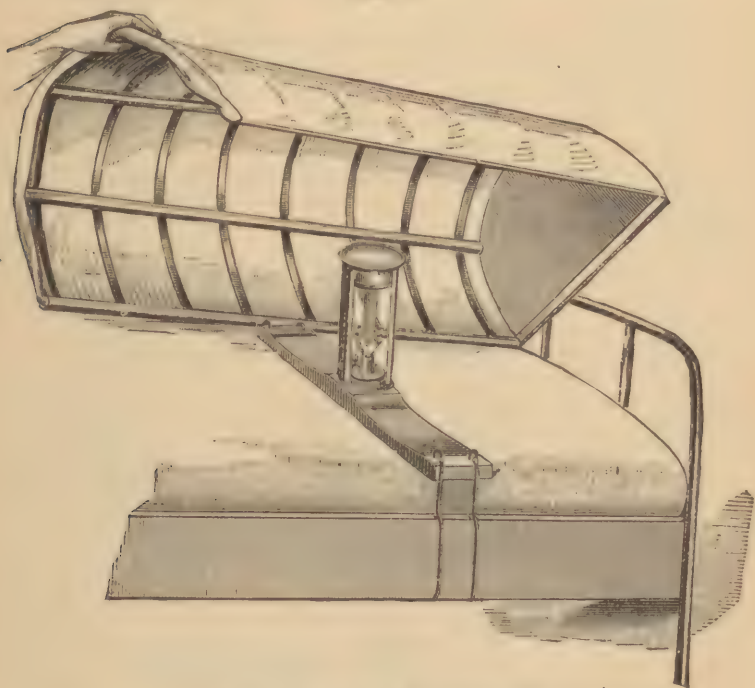
Subcutaneous injections of pilocarpine may also be employed as a



diaphoretic, and in my hands they have produced very satisfactory results. After daily injections (0.1 : 10, one syringe-ful subcutaneously) and the subsequent profuse sweats the exudation was absorbed with surprising rapidity. Before the injection and during the diaphoresis, strong wine or brandy should be given freely, in order to prevent emesis and also to avoid sudden prostration. In one case I obtained a surprisingly favorable effect from the administration of salicylate of soda (six doses daily of 0.5 every fifteen minutes). This was followed by very profuse perspiration and rapid absorption of a long-standing exudation. But this remedy should be watched even more carefully than pilocarpine because there is great danger of sudden collapse. The drug should be given under our personal supervision.

It may here be mentioned that in one case Schmidt observed a serous

FIG. 96.



Sweat-box with auxiliary apparatus.

pleurisy disappear in a few days after erysipelas of the integument of the thorax had developed as the result of the application of a blister.

In anæmic, feeble individuals, absorption is often aided by nourishing diet and tonics. The milk cure is advisable in many cases. The patients take nothing but well-skimmed milk, beginning with 60-180 three times a day and gradually increasing the quantity. It should be taken slowly and in small mouthfuls. Preparations of iron and quinine are also indicated.

According to one plan of treatment, the patient abstains from fluids as much as possible in order to "thicken" the blood and thus facilitate

absorption. Favorable results have been reported, but the plan is attended with too much inconvenience to meet with much success.

It should here be mentioned that a physically demonstrable diminution in the height of the exudation must not always be regarded as the result of absorption. If the dilatation of the affected side of the thorax has increased or if the adjacent organs have been displaced to a greater extent, the upper level of the exudation may be lower than before, although its amount is unchanged or is even increased. It is only when the possibilities referred to have been excluded, that a sinking of the upper level of the exudation may be attributed to absorption of the fluid. It should also be borne in mind that diuresis increases when absorption occurs. I have repeatedly observed that in cases which had previously run an apyrexial course, and in which absorption occurred to a certain extent in spells, short and apparently spontaneous attacks of fever suddenly occurred which could be explained in no other way than as fever due to absorption.

If, in a serous exudation of considerable amount, absorption does not make headway by the end of the fourth week or beginning of the fifth week, or if the exudation then increases in amount, or if its size is dangerous from the start, the fluid must be removed by puncture of the thorax. If the exudation returns, the puncture must be repeated as soon as the above-mentioned conditions obtain. Air must be entirely excluded during the operation, since the introduction of infectious germs into the pleural cavity would give rise to a purulent or even ichorous inflammation. For this reason, also, the instrument employed must be kept scrupulously clean.

If a simple trocar is used, the fluid will not escape, unless the pressure within the pleural cavity is greater than that of the external air. As a rule, this holds good, but exceptions are sometimes observed.

Among Leyden's cases the greatest pressure in the pleuritic exudation was 28 mm. of mercury. In a number of cases the pressure was zero, and Fraenkel and Schreiber report a case in which the pressure within the pleural cavity was negative ( $-6$  Mm. Hg.). Under the latter condition, the introduction of an ordinary trocar would be attended with danger, inasmuch as the external air would be aspirated into the pleural cavity.

Even if the conditions are favorable to the use of a simple trocar, the

FIG. 97.



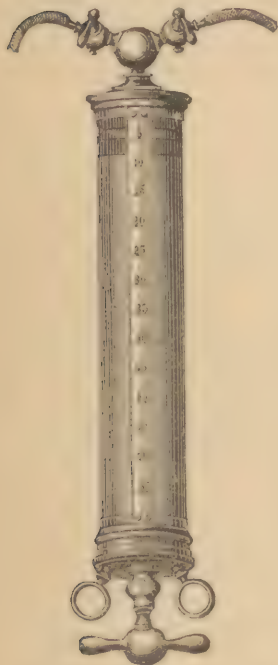
Simple trocar with stop-cock.

fluid would continue to escape only until the pressure within the pleural cavity had become equal to that of the external air, so that the effect of the puncture may be entirely insufficient. In addition, there is the further danger that, after the equalization of pressure has been effected, sudden deep respirations or coughing movements would cause the pressure in the thorax to become negative, so that aspiration of the atmospheric air would be inevitable. Hence puncture of the thorax with a simple trocar is bad practice.

Among the methods adopted to prevent the entrance of air into the chest when employing the ordinary trocar, a very simple and effective

one is the use of a moistened condom. The trocar is pushed through the condom and the latter fastened with threads to the posterior end of the canula. After the armed trocar has been passed into the pleural cavity, the condom is drawn over the free opening of the canula while the stilet is being removed. If the escape of fluid ceases, or negative pressure develops within the pleural cavity, the thin walls of the condom become applied to one another in front of the opening of the canula, and the entrance of air into the pleural cavity is thus rendered impossible.

FIG. 98.



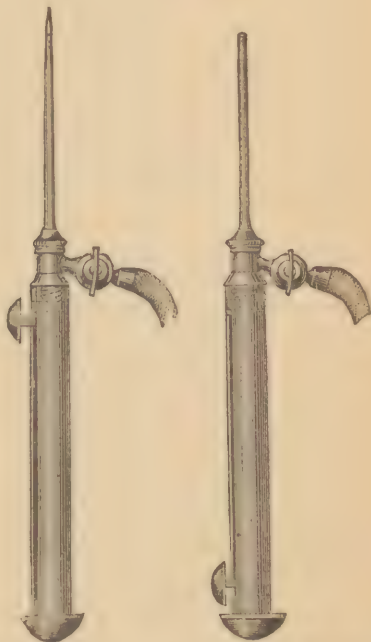
Aspiration syringe, with double stop-cock, for removal of pleuritic exudation.  $\frac{1}{2}$  natural size.

FIG. 99.



Aspirating needle.

FIG. 100.



Fraentzel's trocar. *a*, with projecting stilet; *b*, with stilet withdrawn.

The following method is also simple and effective: A trocar is employed, provided with a stop-cock (Fig. 97) at a short distance from the free end of the canula. A mark upon the stilet shows when its anterior tip passes the stop-cock as the stilet is withdrawn. The trocar is introduced and the stilet drawn back as far as the mark. The stop-cock is then closed and the stilet removed from the canula. A rubber tube is then fastened to the free end of the canula, the other end of the tube being placed in a vessel of water. If the stop-cock is now opened, the free escape of fluid from the pleura is permitted, but the entrance of air is impossible, because the end of the rubber tube is under water.

But when either of the methods described is adopted, the escape of fluid ceases as soon as the pressure within and without the chest has become equal. Hence it is always better to employ aspiration.



A siphon arrangement is often employed for this purpose. A trocar, provided with a stop-cock, is introduced, the stilet removed, and the canula armed with a long rubber tube, which is then filled with disinfected water. The stop-cock is then opened, and as the free end of the tube is depressed, the fluid will begin to flow from the pleural cavity.

The simplest and most serviceable apparatus is a syringe provided with a double stop-cock. The syringe is connected in front with two tubes which either start separately from the bottom of the syringe or have a common attachment. In the latter event, the cavity of the syringe, by means of a stop-cock provided with two canals, may be connected with one tube or the other, according as the stop-cock is turned. But the arrangement is such that, when the cavity of the syringe connects with one tube, it is thrown out of connection with the other. Hence it is possible to draw in the fluid through one tube and discharge it through the other. In our opinion, it is preferable to have a separate stop-cock for each tube (Fig. 98).

The trocar described above may be employed to connect the canula of the syringe with the pleural cavity. After the trocar is introduced, its canula is connected by a rubber tube with one tube of the syringe; the stop-cock of this tube of the syringe is then opened, that of the other tube closed, and the piston is slowly drawn out. Then the stop-cock of the tube connected with the pleura is closed, the other opened, and the fluid is discharged. This operation is repeated until the pleural cavity is emptied.

In order to avoid all chance of entrance of air into the chest, the tube through which the fluid is discharged from the syringe is connected with a rubber tube, the end of which is kept under water. In addition, a little of the aspirated fluid may be retained in the syringe.

The trocar employed to enter the thorax should have a small calibre, or we may make use of a hollow needle, similar to those which form part of a hypodermic syringe (Fig. 99).

If the exudation is very flocculent, large flakes may be applied to the inner opening of the canula, or be aspirated into its lumen, so that it becomes impermeable. The needle must then be removed and introduced into another part of the chest.

Fraentzel has devised a very serviceable trocar with the object of removing any obstruction in the canula (Fig. 100). It consists of a stilet which, by means of a lateral screw, can be moved to and fro in a narrow (so-called capillary) canula. The stilet having been pushed forward, the instrument is introduced into the chest, the stilet drawn back, and the fluid then aspirated in the ordinary way. If the canula becomes obstructed, the stilet is pushed forward and the obstruction thus removed in a mechanical manner.

We will also give a short description of Potain's apparatus (Fig. 101). This consists of a trocar (*a*) which can be elongated by means of small screw arrangement (*b*). Into the latter is screwed a tube (*c*), with a stop-cock and lateral overflow tube. The lateral tube may be connected by means of the tube with the rubber stopper (*f*), which is firmly inserted into a bottle, and is provided with two stop-cocks. The other stop-cock of the stopper is connected by means of the tube (*g*) with the aspiration syringe (*h*). The instrument is used in the following manner: The stop-cock leading to *e* is closed, that leading to *g* is opened, and the air in the bottle rarefied by means of the air-pump *h*. When the piston of the syringe can no longer be moved with facility, *f* is closed. The trocar is

now inserted into the thorax, the stilet is cautiously withdrawn until arrested, and the stop-cock *e* is closed. If the stop-cock in *f* belonging to the tube *e* is now opened, the pleuritic exudation will be drawn into

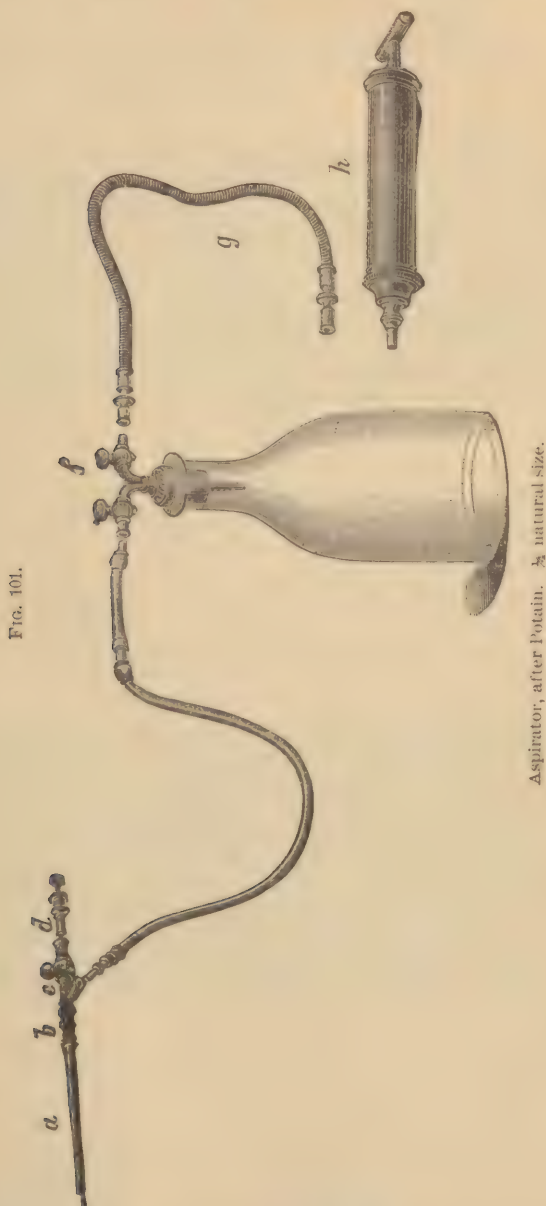


FIG. 101.

Aspirator, after Potain.  $\frac{2}{3}$  natural size.

the bottle. When the bottle is nearly full, the stop-cock connected with *e* is closed, the bottle emptied, and the operation is then repeated.

Age offers no contra-indication to aspiration, and I have repeatedly

performed the operation in children two or three years old. Nor is the existence of fever a contra-indication. Even if fever is present, it almost always disappears or diminishes after the operation.

During the operation the patient should assume a position midway between the horizontal and vertical position, and at the same time midway between complete lateral and dorsal decubitus upon the healthy side. He should be brought close to the edge of the bed, so that the greater part of the thorax is readily accessible. As a general thing, it is advisable to show the patient the trocar or hollow needle, and to explain that the operation is merely equivalent to the entrance of a needle into the skin. The most favorable site of puncture is usually the space between the posterior axillary line and the scapular line. The intercostal space selected should be as low as practicable, taking care that the liver or spleen is not injured. The aspiration should be effected slowly, and at intervals it should be stopped altogether. In removing the canula, the skin should be firmly pressed between the thumb and index-finger against the canula, and the opening covered with adhesive plaster.

We should not remove the greatest possible amount of fluid from the pleural cavity at the operation. Experience teaches that even if small quantities are removed, the remainder is very often absorbed spontaneously with surprising rapidity. This is probably owing to the fact that, as a result of the very marked pressure upon the thoracic walls, the vessels which are engaged in absorption are compressed, and this pressure is relieved—and hence absorption may occur—when a portion of the exudation is removed.

Theoretical considerations also convince us that complete discharge of the serous fluid is not desirable. If the evacuation of the fluid were complete, a *restitutio ad integrum* would only be possible if the lungs were perfectly movable and dilatable, and the previously distended thorax had returned to its normal position. If these conditions do not exist, it is evident that a space will be formed in the pleural cavity, with an abnormally high negative pressure, and which, as a matter of course, will affect the circulatory conditions of the lungs. The lung adjacent to the space containing rarefied air is in a condition as if it were under a dry cup. Under these circumstances, there is danger that excessive dilatation of the pulmonary vessels will give rise to œdema, and this may prove fatal. This is more apt to occur the longer the exudation has been present, and hence offers another reason for early puncture.

The amount which should be removed by the operation depends on individual circumstances, particularly on the ease with which the fluid follows the aspiratory force of the syringe. The elasticity of the thorax, and the dilatability of the lungs must also be taken into consideration. Aspiration should be discontinued at once if a purely serous fluid begins to grow bloody, if violent cough and severe pains make their appearance, or if the lung is felt to rub upon the tip of the canula. After aspiration, the pulse is usually found to be fuller, and its rapidity may be diminished nearly a half. This is also shown by the sphygmographic tracings (Fig. 102 and 103). Diuresis is also increased. Laboulbène and Bonneville found slight rise of temperature in the rectum.

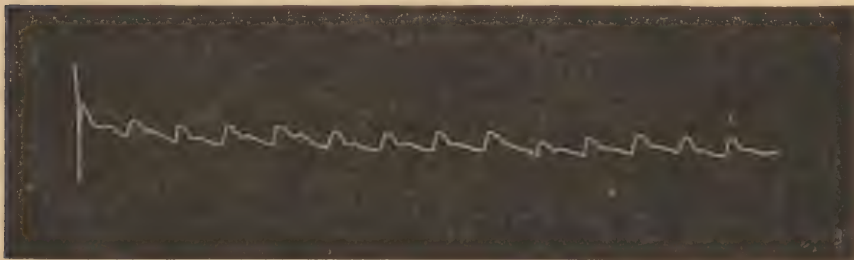
After aspiration, we may await further developments for ten days or two weeks. If the conditions are unchanged, or the fluid again increases in amount, aspiration may be repeated.

Aspiration is rarely complicated by accidents. Syncope may occur,



and presents little significance if it is the result of fright. Very dangerous syncope (anæmia of the brain) may also occur from the sudden change in the circulation of the blood caused by the reopening of the lung to the circulation. This accident is so much more apt to occur the more rapidly the fluid has been removed. Sudden death may result from this condition. Aspiration must be stopped immediately, the head of the patient placed as low as possible, and external and internal irritants applied

FIG. 102.

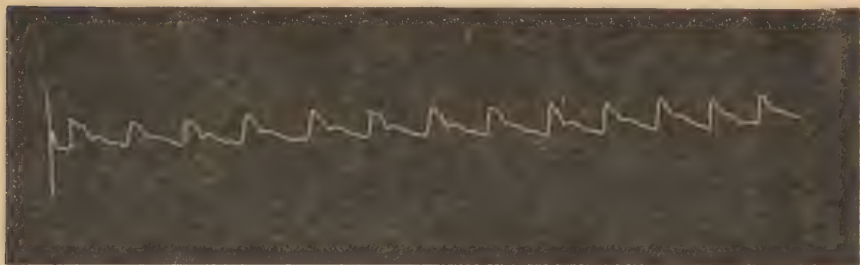


Pulse curve of the right radial artery in serous pleurisy, in a man æt. 20 years, immediately before aspiration.

(inhalation of ammonia, rubbing the temples with brandy or vinegar, brushing the skin, wine internally, etc.). In certain cases, the sudden death seems to be of embolic origin; for, if thrombi have developed between the trabeculae of the heart or in the pulmonary veins, they are readily loosened after puncture has changed the circulatory conditions, and are carried off as emboli.

Pneumonic processes occasionally develop in that portion of the lung

FIG. 103.



The same, shortly after aspiration (a little more than 2½ litres of fluid were removed).

which had been compressed, and are usually of a very flaccid character (so called serous pneumonia). In one case, Fraentzel observed a fatal pulmonary hemorrhage from the vessels of a cavity which was situated in the compressed lung.

Empyema requires operative treatment as early as possible. Every day that is lost makes the prognosis more serious. Almost all surgeons agree that incision alone should be performed.

When aspiration is employed in empyema, the exudation returns in the majority of cases, and the patient's strength is thus frittered away. Puncture offers the greatest chances in the empyema of children, especially if the pleurisy has not lasted for a long time. It has recently been

recommended that a portion of the pus be removed by aspiration, the remainder then diluted by the injection of an antiseptic fluid into the pleural cavity, and the mixture then removed by aspiration. This procedure is to be repeated until the pleural cavity has been washed clean. We have no doubt, as has been shown by some favorable reports, that recovery may be secured in this way, but we do not consider this plan of treatment reliable.

Puncture in empyema not alone wastes time and strength, but a number of cases have been reported in which it was followed by pneumothorax.

Empyema operations by incision would undoubtedly give better results if the operation were always done by experienced surgeons, and if it were made at once after the exploratory puncture.

Age furnishes no contra-indication to the operation. I have seen a number of children at the age of two or three years recover rapidly after the operation.

The incision should be made while the patient is under the influence of chloroform. We should not be satisfied with a simple incision in the intercostal space, since the wound usually closes before recovery is complete. Surgeons will agree that the resection of a portion of a rib does not materially complicate the operation, and as this alone prevents the premature closure of the wound, a piece of the rib should first be excised, and the incision into the costal pleura made at this place. The best site for the operation is the axillary region from the fifth to the eighth ribs.

v. Langenbeck attempted trephining instead of resection of a rib, but it has been objected that the trephine opening is often too small, and that the bone is apt to fracture.

In multilocular empyema, the incision may open only into one compartment. Sometimes an adjacent compartment ruptures spontaneously, or we may be compelled to make a second incision.

In one case, Darwin observed that, despite two incisions, the pus ruptured into the lung and also externally.

Serious accidents also occur, though very rarely, after the operation for empyema. We refer to attacks of syncope, convulsions, choreiform movements, monoplegic and hemiplegic symptoms, either with or without an anatomical basis. In the latter event, the lesion is usually embolism of the carotid and cerebral arteries, dependent on thrombosis of the pulmonary veins or left auricle. Similar symptoms have been observed during the washing of the empyema cavity, perhaps because the stream of water impinged directly upon the heart and lungs, and loosened thrombi situated within them. The paralysis sometimes disappears so rapidly that it must be regarded as reflex. Hyperidrosis and vaso-motor disturbances have also been observed.

I know of one case in which erosion of an intercostal artery and death from hemorrhage occurred, despite the fact that incision had been performed.

The operation should be performed in strict accordance with Lister's methods. It then becomes unnecessary to wash out the pleura; at the most, it may be cleansed immediately after the incision with a lukewarm solution of salicylic acid. A drainage tube is placed in the wound, and upon changing the dressing, the lower limbs of the patient should be elevated so that all the secretion may flow from the tube. I have seen a number of cases recover entirely in a surprisingly short period under this plan of treatment.

If empyema necessitatis has developed, the same mode of treatment is indicated, except that the incision must be made at the site of the prominence produced by the pus.

Even after the empyema has perforated into the lungs, the operation becomes necessary if expectoration ceases, if the development of hectic fever indicates the stagnation of pus, and if the amount of fluid is found to remain unchanged. If the excision must be followed by washing out of the pleural cavity on account of putrid decomposition of the pus, this should be done very cautiously because, on account of the open communication between the pleural cavity and the lungs and bronchi, the latter may be inundated with the pus and fluid, and the patient thus put in danger of suffocation.

The question of operative interference in pleurisy occurring in phthisical patients has been widely discussed. With regard to empyema, our experience leads us to answer the question with a decided affirmative. In serous pleurisy, likewise, we think the operation should be performed if the amount of exudation endangers life; if not, we may await events.

The treatment of putrid pleurisy is very similar to that of empyema. An incision should be made and at each dressing the pleural cavity should be thoroughly cleaned with antiseptic fluids until the foul odor has disappeared. Carbolic acid should not be employed or, at least, this should be done very carefully, because poisoning and rapid death from collapse may be the result of excessive absorption of this substance. For this reason solutions of boracic acid (2-10%), thymol (0.1%), chloride of zinc (3-6%), salicylic acid preparations, and especially acetate of alumina (1-2%) are the most suitable. Since putrid pleurisy is usually the result of pulmonary gangrene, the gangrenous focus in the lungs should be disinfected at the same time (vide page 329). Large shreds of necrotic lung tissue occasionally make their exit through the site of incision.

Hemorrhagic pleurisy is almost always the result of some general disease. Local treatment should be delayed as long as possible, and surgical interference withheld until the amount of fluid proves a source of danger.

Certain symptoms of pleurisy sometimes become so prominent that they demand our chief attention. This may be true of the fever. High fever should be treated by enemata of antipyrin (4.0-6.0 in 2 ounces of lukewarm water). In addition, large doses of alcohol are necessary in order to prevent exhaustion. The local application of ice to the affected side of the chest will also be useful. Treatment with antipyrin, cold baths, and stimulants is stringently indicated in pleuritis acutissima.

If the cough is severe, mild narcotics are indicated, perhaps associated with expectorants.

Pulmonary gymnastics should be resorted to if retraction of the thorax remains after the absorption of the fluid. The patient should raise the arm on the affected side and take deep respirations several times a day. At first these exercises should be short and then gradually prolonged. The inhalation of compressed air seems to us to be less rational, since there is danger of excessive dilatation of the healthy lung.

Retraction of the thorax has also been relieved in a number of cases by resection of the ribs [vide the text-books on surgery].

Thoracic fistulae require the treatment recommended in empyema.



## 2. *Pneumothorax. Hydropneumothorax.*

I. **ETIOLOGY.**—Pneumothorax means an accumulation of air in the pleural cavity. In addition to air, the pleural cavity usually contains fluid, so that hydropneumothorax is more frequent than pneumothorax. According as the fluid is serum, pus, or blood, the disease is known as sero-pneumothorax, pyopneumothorax, hamopneumothorax.

An accumulation of fluid, especially of pus, is sometimes found at the beginning and is followed by pneumothorax. In other cases the air first enters and gives rise secondarily to exudative pleurisy. Under certain circumstances, for example the perforation of a cavity through the pleura, gas and fluid may enter the pleural cavity at the same time.

Pneumothorax and hydropneumothorax are either diffuse (free) or sacculated and circumscribed. In the large majority of cases, the disease is unilateral. It is sometimes found that pneumothorax recurs, after a certain period, upon the same side of the chest. The disease is either acute or chronic.

The conditions for the development of pneumothorax are always furnished when a space filled with air enters into communication with the pleural cavity. Other causes are unknown.

Some writers believe that pneumothorax may be the result of the decomposition of a purulent exudation in the pleural cavity, but other writers deny the possibility of such an occurrence. Recent analyses of the gases do not favor the theory of the spontaneous development of the gases, but there are a number of cases in which careful search failed to discover the communication between a pyopneumothorax and some other air-containing space.

Pneumothorax develops most frequently in the course of pulmonary diseases, especially phthisis. The phthisical degeneration extends to the pulmonary pleura, so that air passes from the lungs into the pleural cavity. The perforation occurs occasionally without any special cause, while in other cases the final perforation is the result of coughing, straining, and similar bodily movements. As a general thing, there is a special tendency to the development of pneumothorax on the part of those pulmonary changes in which destruction proceeds rapidly, while in ulcerative processes which run a chronic course, thickening of the pulmonary pleura often occurs, or fibrous adhesions form between both layers of the pleura. These changes combat the threatening perforation, and produce a sort of natural recovery. Perforation of the pulmonary pleura rarely occurs at the apex; it is usually found at the lower border of the upper lobe, or the upper border of the middle lobe, more rarely in the upper portions of the lower lobe. The most frequent site is the region between the mammary and axillary lines at the level of the third and fourth intercostal spaces.

In view of the frequent phthisical origin of pneumothorax, it is not surprising that the latter is more frequent on the left side than on the right, since phthisical changes in the left lung have a greater tendency to progressive destruction than those in the right lung. Powell observed pneumothorax in five per cent of his phthisical cases, and among seventeen cases of the former disease, ten occurred on the left side.

Not alone large cavities situated beneath the pulmonary pleura, but likewise small peripheral, cheesy nodules may give rise to pneumothorax. These are occasionally so small that they escape physical diagnosis, and the etiology is only explained by the autopsy.

All other pulmonary diseases which lead to ulcerative processes in the lungs may give rise to pneumothorax. This category includes pulmonary abscess, gangrene, and occasionally peripheral bronchiectasis. Echinococci, which are situated immediately beneath the pulmonary pleura, may also give rise to the disease, if the vesicle ruptures and bursts at the same time into a bronchus and the pleural cavity.

In certain cases pneumothorax is the result of injury to the lungs, either direct or indirect.

Stab-wounds, gunshot wounds, etc., may injure both the chest-wall and the lungs, and, while the external injury may be of such a nature that it does not allow the entrance of air into the pleural cavity, the pulmonary wound gives rise to pneumothorax. Niemeyer claimed that this usually obtains. If the ribs are fractured by the injury, the thoracic wall may not be opened, but the sharp ends of the ribs may penetrate the pleura and lungs, and thus give rise to pneumothorax. The lungs may also be ruptured as the result of traumatism, and pneumothorax may be produced, although the ribs and chest wall are uninjured. Sée states that the escape of air from the lungs sometimes occurs at the site of injury, sometimes opposite to this situation as the result of contrecoup.

Closely related to these cases are those in which rupture of the lungs and pneumothorax are the result of violent bodily exertion, coughing, and straining. Not a small number of such cases have been reported. The accident has also been observed in whooping-cough and asthma. Finally, a case has been reported in which forced coitus gave rise to pneumothorax.

All these conditions are especially dangerous when they occur in persons suffering from pulmonary emphysema, in whom peripheral alveolar ectases have a tendency to rupture. Indeed, it would seem as if progressive atrophy and thinning of such ectases may give rise to spontaneous rupture. Bajasinski reports a case in a man suffering from emphysema, and in which pneumothorax developed during sleep, and was not preceded by cough.

The disease sometimes occurs in empyema when the latter perforates the lungs or the walls of the chest. Not every perforating empyema gives rise to pneumothorax, because the often fistulous site of perforation may have a valve-like structure which permits the escape of pus, but does not allow the entrance of air into the pleural cavity. Pyo-pneumothorax will also be produced if the empyema perforates any other organ filled with air (bronchi, trachea, œsophagus, stomach, intestines).

Injuries to the thorax necessarily give rise to pneumothorax when they permit the entrance of air. In simple stab-wounds, this is by no means frequent, and Wintrich showed by experiments on animals that it is very difficult to produce pneumothorax in this manner. Abscess, gangrenous processes, ulcerating cancers of the chest-walls, may also give rise to pneumothorax.

This malady is also an occasional result of disease of certain of the mediastinal organs, for example, suppuration of the bronchial glands, which rupture at the same time into the bronchi and pleural cavity. In other cases, abscesses and cancers of the œsophagus perforate the pleural cavity. Furthermore, in the careless use of the œsophageal sound the latter may enter the pleural cavity and produce pneumothorax.

In another series of cases, it is the result of diseases of the abdominal organs. For example, in cancers and ulcers of the stomach and intestines, if the latter have become adherent to the diaphragm

and this undergoes perforation. Oppolzer also states that he has seen softening of the stomach and diaphragm, and perforation of the latter during the course of typhoid fever. In like manner, every abscess and ulcerative process in the abdominal cavity may give rise to pneumothorax, as soon as a communication has been formed between the pleural cavity and the stomach or intestines.

The following table gives the causes of pneumothorax in 918 cases collected by Biach:

Pulmonary phthisis, . . . . .	715
Pulmonary gangrene, . . . . .	65
Empyema, . . . . .	45
Traumatism, . . . . .	32
Bronchiectasis, . . . . .	10
Pulmonary abscess, . . . . .	10
Emphysema, . . . . .	7
Gangrenous hemorrhagic infarction, . . . . .	4
Thoracocentesis, . . . . .	3
Perforation of the œsophagus, . . . . .	2
Perforation of the stomach, . . . . .	2
Round worms in the pleural cavity, . . . . .	2
Echinococci of the lungs, . . . . .	1
Perforation of an encapsulated peritoneal exudation, . . . . .	1
Perforation of bronchial glands, . . . . .	1
Caries of the ribs, . . . . .	1
Caries of the sternum, . . . . .	1
Abscess of the mammary gland, . . . . .	1
Fistula between pleura and colon from hydatids, . . . . .	1
Unknown causes, . . . . .	14
<b>Total, . . . . .</b>	<b>918</b>

Lentz gives the following table of 35 cases occurring in childhood:

Pulmonary phthisis, . . . . .	14
Pulmonary gangrene, . . . . .	11
Emphysema, . . . . .	3
Pulmonary apoplexy, . . . . .	3
Fracture of the ribs, . . . . .	1
Empyema, . . . . .	1
Bronchiectasis, . . . . .	1
Hemorrhagic infarction, . . . . .	1
<b>Total, . . . . .</b>	<b>35</b>

Cases of pure pneumothorax are rare. As a matter of course, it can only be looked for when air enters a previously intact pleural cavity; but even under such circumstances hydropneumothorax generally develops. In the latter event, the air itself does not act as an inflammatory irritant, but this effect is produced by the organized germs contained in it. If the air happens to be free of schizomycetes, simple pneumothorax will be produced. This occurs most frequently when the disease is the result of emphysema or of a solution of continuity of the lungs, as the result of sudden, excessive bodily exertion.

After hydropneumothorax develops, it is found not infrequently that the air is absorbed and a simple exudative pleurisy remains. In the majority of cases, the pleural cavity contains pus in addition to the air. Cases of seropneumothorax are much rarer, though they are not so uncommon as is generally believed. In rare cases, we find hæmopneumothorax, especially after rupture of the lungs.

The structure of the air fistula exerts a remarkably great effect upon the clinical and anatomical features of the disease. Weil recognizes four



varieties of pneumothorax, viz.: open, valvular, and closed pneumothorax, and a transition form between these three.

In open pneumothorax there is a gaping opening which allows the free passage of air. The opening is slit-shaped, round or irregular. In phthisical cavities I have several times found round openings in the pulmonary pleura, almost as large as a "mark" piece. Under such conditions, the air enters the pleural cavity until the pressure within the latter is equal to that of the atmospheric air.

A special form of open pneumothorax is that in which there is one opening in the lung, another through the chest-walls as, for example, when an empyema, which has perforated the lung, is operated upon by incision.

In valvular pneumothorax, the valve may be of such a character that air is admitted into the pleural cavity during inspiration alone, and cannot be expelled during expiration. Under such circumstances, the pressure within the pleural cavity may exceed that of the external air. The further entrance of air will cease only when the pressure has reached its maximum, or when the fistula is closed by the pressure itself, or when the fistulous opening closes spontaneously. It is evident that the greater the pressure within the pleural cavity, the greater will be the compression and displacement of adjacent organs.

A closed pneumothorax is one in which no communication is possible between the air in the pleural cavity and the atmosphere, whether the fistula is closed or open. The pressure in the pleural cavity and the symptoms of compression and displacement depend exclusively upon the amount of air in the pleural cavity.

The transitional forms constitute an intermediate or secondary stage. Thus, a valvular pneumothorax may be converted into an open or closed pneumothorax, etc.

II. ANATOMICAL CHANGES.—On autopsy, the affected side is sometimes found inordinately distended, but this may be less marked than during life, because the tension of the gases has diminished. If the pressure of the gas is increased, it will escape with a hissing noise upon making an incision in the chest. If the site of incision is kept under water, bubbles of gas will be seen rising through the water. The escaping gas may be entirely inodorous, or it may smell like sulphuretted hydrogen. Its principal constituents are nitrogen, oxygen, carbonic acid, and traces of marsh gas.

In the majority of cases, the pleural cavity also contains exudation. As a rule, this consists of pus, more rarely of serum or blood. The pus is rarely decomposed in cases of open pneumothorax.

In free pneumothorax the lung is collapsed and not infrequently compressed into a non-aërated mass situated near the spine, and immediately adjacent to the mediastinum. The heart, diaphragm, and liver or spleen are displaced, except in cases of old and firm adhesions.

The site of perforation may be recognized at once in many cases of open pneumothorax. If not, a tube should be placed in the bronchus, and the lung, which is kept under water, should be distended. We must then look for the place whence the air bubbles rise. The site of perforation is not always easy to determine, on account of the presence of adhesions and new membranes.

III. SYMPTOMS.—If pneumothorax develops suddenly while the respiratory organs are comparatively unaffected, the symptoms of dangerous dyspnœa suddenly make their appearance. The patients sometimes

fall down unconscious, or they cry aloud that something has burst within the chest. Some patients make the statement that they experienced the sensation of an ascending current of air within the chest. The features express indescribable fear, and the face is often covered with a cold sweat. The limbs feel cold; the pulse is small and frequent, the respirations are labored and accelerated. Cyanosis is extreme.

If the pneumothorax is the result of phthisical changes in the lungs, careful examination alone may reveal the occurrence of the accident, so slight are the symptoms which may be produced thereby. At all times the diagnosis can only be made positively after physical examination.

Upon inspection, the affected side of the chest is found to be dilated in free pneumothorax, sometimes to the extent of five to eight cm. The intercostal depressions are usually obliterated, sometimes they even project outwardly. The respiratory movements are either absent or very slight upon the affected side. In the latter event they are irregular and jerky.

Pneumothorax does not occur when the chest is retracted except in cases of empyema. When complicated with hydropneumothorax, the dilatation of the thorax may increase still further, although the air is gradually absorbed.

Very striking phenomena are produced not infrequently by the displacement of the heart and liver. In left pneumothorax the heart is pushed into the right thorax, and is occasionally seen pulsating to the outside of the right mammary line. At the same time the liver is pushed downwards to a greater or less extent, so that the hepatic region becomes very prominent, or the lower border of the organ appears beneath the abdominal walls as a transverse prominence which moves on respiration. In right pneumothorax the apex beat is visible outside of the left mammary line, not infrequently in the left axillary line. The degree of displacement depends not alone on the amount of air in the pleural cavity, but also upon the mobility of the adjacent organs.

The patients almost always maintain a passive position of the body. They lie constantly on the affected side in order to facilitate the respiratory movements of the unaffected lung. In phthisical patients, in whom pneumothorax has developed insidiously, a suspicion of the accident should be aroused when the patient suddenly assumes and maintains the lateral decubitus. Some patients assume a sitting and orthopnoëic position.

In cases of open hydropneumothorax, the patients sometimes assume very remarkable positions if they find that the expectoration and discharge of the fluid are thereby facilitated. In Romberg's often cited case the patient assumed, at intervals, the dorsal position with the head hanging very low, because in this position he could cough up large amounts of fluid from the pleural cavity. In this case the unclosed fistula was situated quite high up. Abdominal decubitus has also been observed in open, but encapsulated pneumothorax of the anterior surface, because the patient thus endeavors to avoid the uninterrupted flow of the fluid to the fistula and into the bronchi, and at the same time prevent the constant cough.

If the pneumothorax develops when the lungs are healthy or slightly diseased, the signs of objective dyspnea will be recognized without difficulty. The respirations are accelerated and labored. The auxiliary muscles of respiration are brought into play, and there is more or less cyanosis.

Vocal fremitus is diminished or abolished in pneumothorax and hydropneumothorax, because the fluid and gas obstruct the conduction of waves of sound from the air passages to the chest-walls.

Pleuritic adhesions act as conductors of the waves of sound, and at their costal insertions fremitus may be maintained or even increased.

The resistance of the thorax is generally increased. In hydropneumothorax, the feeling of resistance is considerably diminished above the fluid, so that the level of the fluid can thus be determined with considerable accuracy.

When the pleural cavity contains air and fluid, a short, vigorous shaking of the patient (succussion) sometimes gives rise to a splashing sound. A feeling of fluctuation is also felt, in some cases, in the intercostal spaces.

The symptoms on percussion vary, and depend chiefly on the tension of the thoracic walls. In open pneumothorax, in which the atmospheric pressure affects both sides of the chest-wall, the percussion sound is strikingly loud and tympanitic, and also presents a metallic quality.

In many cases the metallic quality is only recognized if the ear is brought close to or in contact with the chest during percussion. In order to produce the metallic sound, Heubner recommends percussion with the handle of the pleximeter, or with its metallic head. Stern recommends percussion with the tip of the nails instead of the soft tips of the fingers. In this manner we hear the metallic sound alone without the tympanitic quality.

If the tension of the chest-wall is very considerable (in valvular or closed pneumothorax), the tympanitic quality of the percussion note is lost, and its intensity is diminished, so that a dull percussion sound is produced. This is analogous to the results obtained in Skoda's experiments, in which it was shown that the stomach, when filled with air, gives a tympanitic percussion sound, but the tympanitic quality is lost when the stomach is excessively distended.

Taube also showed that excessive tension of the chest-walls may render indistinct or abolish the metallic quality of the percussion sound, but that this can be reproduced in the dead body when, as the result of cooling, the tension of the gases, and therefore of the chest-walls, is diminished. It may again be destroyed by pressing the diaphragm upwards, thus increasing the tension of the gases.

So long as the external or internal fistula remains open, cracked-pot resonance is heard on short and vigorous percussion.

This is owing to the fact that at each impulse of percussion the pressure of the air in the pneumothorax is increased, and it therefore passes abruptly through the open fistula. A whirl of air, which gives rise to the hissing sound, is produced upon the other side of the fistula. In percussion of the posterior surface of the thorax, the sound is not conveyed in many cases to the ear of the examiner, who is standing behind the patient. It is well, in such cases, to have percussion performed by another, the physician placing his ear in front of the patient's widely-opened mouth (this manœuvre assists the conduction of the sound to the outside). The cracked-pot resonance of pneumothorax disappears when the fistula closes.

Changed pitch of the percussion sound is sometimes heard in pneumothorax. If the fistula is external, the pitch always becomes lower if the opening is closed with the fingers. If there is a large fistula in the pulmonary pleura, the pitch may become higher on opening the mouth,



lower on closing it. If the latter phenomenon has been present, and then disappears, we have evidence that the fistula has closed.

Very noteworthy symptoms are observed on percussion if gas and fluid are both contained in the pleural cavity. The chief characteristic is the change of phenomena on changing the position of the body, because the fluid always tends to assume the lowest position.

In dorsal decubitus, the upper border of dulness is higher posteriorly near the spine than it is anteriorly. In the sitting position, the upper border is a horizontal line; in lying upon the healthy side the lateral dulness may disappear entirely.

Gentle percussion should be employed in percussing the upper level of the fluid. If this is not done, the dulness begins too low, and the amount of fluid appears smaller than it really is. Palpatory percussion may also be employed, the increasing feeling of resistance indicating the level of the fluid.

Change in the position of the body sometimes changes the pitch of the metallic percussion sound. Biermer found that, in the sitting position, the pitch becomes lower, because the fluid then pushes the parietic diaphragm downwards, and thus increases the largest diameter of the pneumothorax. Bjoernstroem and Weil have observed increased pitch in the sitting position, because the amount of fluid, the resistance of the diaphragm, and the shape of the pleural cavity may cause a shortening of the greatest diameter of the pneumothorax. Bjoernstroem also noticed that the pitch of the metallic percussion sound was a little higher during inspiration than during expiration. If the pneumothorax is situated near the heart, the pitch may vary with each cardiac contraction.

The pitch of the metallic percussion sound also varies as the amount of fluid increases or diminishes, and thus forms a valuable sign in diagnosis.

Cracked-pot resonance may also be found in hydropneumothorax, but only when the open fistula is situated permanently above the upper level of the fluid or is made free in certain positions of the body. The latter circumstance aids us in an approximate determination of the position of the fistula.

The succussion sound, which is heard in hydropneumothorax alone, is one of the principal auscultatory signs. If the patient is grasped by the shoulders and vigorously shaken to and fro, a metallic splashing sound is heard. Its intensity varies. In some cases it is heard only when the ear is applied to the chest, in others it may be heard the length of the room.

In rare cases the sound is produced by the movements of the heart. The succussion sound is not produced exclusively in hydropneumothorax. The conditions for its development are the presence of air and fluid in a large, smooth-walled cavity. Hence it may be heard in pulmonary cavities. But it is heard with such overwhelming frequency in hydropneumothorax that it possesses a certain degree of pathognomonic significance.

The sound of falling drops is also heard occasionally in hydropneumothorax. In one case Leichtenstern attempted to show that it was really the result of falling drops. It was heard in the patient as soon as he changed from the recumbent to the sitting position. On autopsy fibrinous prolongations and adhesions were found upon the pleura, and those which dipped beneath the fluid during dorsal decubitus, allowed the fluid to drop when the patient assumed the sitting position.

The auscultatory signs vary in pure pneumothorax. In some cases the respiratory murmur is absent, and it has been maintained that a very marked unilateral distention of the thorax, when associated with absence of the respiratory murmur, should arouse the suspicion of pneumothorax. In other cases the respiratory murmur has an amphoric or metallic quality.

The metallic sounds are usually loudest in the interseapular space, because here, as a rule, the lung is in closest contact with the thoracic walls. It is sometimes heard only during expiration, in other cases during both phases of respiration, and may then be louder during inspiration than during expiration. The distinctness of metallic breathing varies greatly in different parts of the chest. The conditions for the production of the metallic respiratory murmur are also furnished when air from the lungs passes through an open fistula into the pneumothoracic space.

If the conditions for the production of râles are present in the lungs, they will possess a metallic quality if transmitted through the pneumothorax. If single râles are produced, they may give rise to the sound of the falling drop.

Metallic auscultatory phenomena are present in hydropneumothorax, but depend upon the pneumothoracic portion of the disease.

Bronchophony is diminished and also possesses a metallic quality.

Pneumothorax is sometimes observed upon one side, exudative pleurisy upon the other side of the chest.

The symptoms of pneumothorax are sometimes confined to the local changes mentioned above. This is observed with relative frequency when large phthisical cavities are present in the lungs.

In other cases certain phenomena point to the development of pneumothorax, although the symptoms may remain purely local. For example, if large masses of pus are suddenly expectorated in the course of an empyema, it may be concluded that the pus has perforated into the lungs, and the patient should be examined for pneumothorax.

Very severe symptoms may arise if the pneumothorax occurs unexpectedly, the lungs and pleura having previously been in a normal condition. In such cases it may act as the immediate cause of death, and may prove fatal in a few minutes.

The patients are tortured by a feeling of intense dyspnoea, cyanosis is marked, and the respiratory movements are labored. At the same time the appearances of serious collapse are presented. The skin is cool and sometimes covered with clammy sweat, the pulse is very frequent and at the same time extraordinarily small, the voice is often aphonic, and the patient talks in a low whisper.

Consciousness may remain entirely intact. In addition to the dyspnoea, the patients complain of violent pain, usually in the inferior part of the chest (probably from excessive tension of the diaphragm and chest-walls).

The cardiac movements are very much accelerated and, on account of the displacement of the organ, the heart sounds are often heard distinctly in abnormally high situations. The heart sounds may present a metallic quality as the result of resonance in the pneumothoracic space.

The peripheral veins are distended. If the pneumothorax continues for a long time, the circulatory disturbances may lead to œdema of the skin, appearing first on the limbs and face. Vertigo, a feeling of weight

in the head, tinnitus aurium, and the like seem to be the effects of circulatory changes in the cerebral veins.

In right-sided pneumothorax, the liver may be depressed to such an extent that its upper border is situated at the lower border of the chest. In left-sided pneumothorax the spleen is pushed downwards and towards the median line and may thus be felt beneath the left hypochondrium.

Diuresis is diminished and traces of albumin are found not infrequently in the concentrated urine. Emphysema of the skin sometimes develops, as the result of entrance of air into the interstitial connective tissue of the lungs or its passage from the œsophagus into the mediastinal connective tissue, and thence into the subcutaneous tissue of the skin.

The signs of pure pneumothorax may persist for several weeks or even months. Hydropneumothorax has been known to last for several years. If the fluid is purulent, waxy degeneration may be produced, and the patient finally dies with symptoms of general marasmus.

IV. DIAGNOSIS.—The diagnosis of free pneumothorax and hydropneumothorax is usually not difficult. The fluid is purulent in the majority of cases, but its character can only be determined with certainty by an exploratory puncture.

The diagnosis of circumscribed pneumothorax is often very difficult; it may be mistaken for a pulmonary cavity, distention of the stomach, diaphragmatic hernia, and pyopneumothorax subphrenicus.

The differential diagnosis between circumscribed hydropneumothorax and superficial pulmonary cavities depends mainly upon two factors. In the first place, the intercostal spaces over a pulmonary cavity are usually depressed; in pneumothorax they are bulging; furthermore, the vocal fremitus over a cavity is increased; over a pneumothorax it is diminished. The diagnosis is especially difficult if change of pitch in the percussion sound is noticed on opening and closing the mouth, because this symptom is characteristic of a cavity. A distinct succussion sound favors the diagnosis of hydropneumothorax, because this sign is exceptional in pulmonary cavities.

If abdominal tympanites, and particularly gaseous distention of the stomach, is very marked, the stomach is sometimes applied so closely to the lower parts of the thorax that the respiratory murmur, râles, and even the heart-sounds acquire a metallic quality from resonance in the organ. In addition, the percussion sound is tympanitic or metallo-tympanitic, and if the stomach contains gas and fluid, a succussion sound may be heard on shaking the patient. The interference with the movements of the diaphragm may give rise to dyspnoea. In making the differential diagnosis, attention should be directed to the clinical history of the disease. Furthermore, the metallic signs connected with distention of the stomach are apt to change rapidly, and they may also be changed by the introduction of fluid into the stomach.

Trentham-Butlin described a case in which pneumothorax was mistaken for diaphragmatic hernia. A man was caught between two cars, and received a contusion of the lower part of the left thorax. This was followed by the signs of left pneumothorax. At the autopsy, it was found that these signs were simulated by a transverse rupture of the left half of the diaphragm, through which the stomach, spleen, and a part of the colon had passed into the pleural cavity. The differential diagnostic data, to which we have referred previously, must be relied upon to avoid similar mistakes.



Under the the term pyopneumothorax subphrenicus, Leyden has described cases in which cavities filled with gas and fluid (pus) are situated beneath the diaphragm, and extend so far into the pleural cavity that they give rise to the signs of true hydropneumothorax. In two cases. Cossy found that the diaphragm was destroyed, with the exception of its pleural lining, so that the diaphragmatic pleura was pushed far into the thorax. This condition develops usually as the result of perforation of the stomach or intestines, following ulcerations, foreign bodies, or injuries. Cases have been reported as the result of perforation of the vermiform appendix and perityphlitis. Leyden has laid down clear rules for the differential diagnosis from true pneumothorax. In many cases, it is decided by the history of the disease, since pyopneumothorax subphrenicus is preceded by perforation peritonitis, while cough and expectoration are usually absent. The lungs may be entirely intact, and present respiratory displacement. The manometer shows that the pressure increases during inspiration, and diminishes during expiration, the reverse holding good in pneumothorax. Schreiber showed, however, that there are exceptions to this rule. Finally, Cossy claims that a faecal odor of the gases discharged on exploratory puncture is indicative of pyopneumothorax subphrenicus.

In many cases of free or sacculated pneumothorax or hydropneumothorax, it may be important to determine whether the fistula is closed or not. It must be regarded as open if cracked-pot resonance is heard on percussion, and also if the pitch of the percussion sound varies upon opening and closing the mouth. In the latter event, it must be assumed that there is a large opening in the pleura. In valvular pneumothorax, however, movement of the air is possible only in one direction, and the movement from the pleural cavity to the air passages is usually interfered with.

Two methods have been adopted in determining the character of the fistula in valvular pneumothorax, viz., analysis of the gases in the pleural cavity, and aspiration of the pleural air.

Ewald found that the presence of more than ten per cent of carbonic acid indicated complete encapsulation of the pneumothorax: if five to ten per cent is present, the closure is imperfect, and if the amount is less than five per cent, the fistula may be regarded as open. Since atmospheric air enters the pleural cavity, the gases within it naturally consist of oxygen, nitrogen, and carbonic acid, but the constitution of the gases varies subsequently according as the fistula is open or not, because the different gases are absorbed by the pleura in different degrees.

If the pneumothoracic space is aspirated in a case of hydropneumothorax, and some of the air is removed, the inclosed air is sometimes rarefied to such an extent that, if the fistula remains open, air will enter from the lungs, and will rise in the fluid with a gurgling sound. As a matter of course, this sound will not be heard if the fistula is closed.

In one case, Riegel heard this sound during inspiration and expiration, and later during inspiration alone, although aspiration had not been performed. When the patient was lifted up, he always expectorated large quantities of pus, on account of which the air in the pleural cavity was rarefied, and air again entered through the open fistula. The murmur during expiration was probably owing to the fact that the level of the pleural fluid sank beneath the fistula, the air from the pleura then passed through the fistula, and formed bubbles in the fluid, which remained in the fistulous opening.

Boisseau states that the metallic signs are much more marked when the fistula is still patent.

If an empyema has perforated and given rise to pyopneumothorax, the character of the sputum and mode of expectoration sometimes indicate the patency of the fistula. The patients expectorate purulent and often very foul-smelling masses, sometimes in "mouthful" expectoration. They may cough only a few times during the day, but remarkably large amounts are expectorated each time. This is owing to the fact that the desire to cough only arises when the cavity is filled with fluid to such an extent that it reaches the fistula and flows into the bronchial passages.

Manometric measurements may be employed to distinguish open and closed or valvular pneumothorax during life. After puncture of the pneumothoracic space with Fraentzel's trocar, the lateral stop-cock is connected with a manometer. If the pressure of the gas in the pleural cavity, during the cessation of respiration, is equal to the atmospheric pressure, the pneumothorax is open. If the pressure exceeds that of the atmosphere, we have to deal with a closed or valvular pneumothorax. If the gas in the pleural cavity is again aspirated in part, a second measurement will show that the pressure is less than it was before aspiration, if the pneumothorax is permanently closed; it remains the same if the pneumothorax is open, and in valvular pneumothorax it continues to increase as the result of spontaneous entrance of air into the pleural cavity, and may exceed the atmospheric pressure. In open pneumothorax, it is generally easy to recognize an internal fistula, in addition to the external one. The patients not infrequently expectorate a fluid which has been introduced into the pleural cavity, and which has either been colored, or contains some substance easily demonstrable on chemical examination. The fluid discharged from the external opening not infrequently contains elastic fibres or other constituents of the sputum.

V. PROGNOSIS.—Under all circumstances the prognosis must be regarded as very serious. The more unexpectedly a pneumothorax develops the greater is the danger to life. As a general thing, the prognosis of open and closed pneumothorax is more favorable than that of valvular pneumothorax, because the latter is apt to be attended with great tension of the gases, and marked displacement of the adjacent organs, especially of the heart.

The prognosis is unfavorable in many cases, because the primary disease offers no hope of recovery.

If the lungs present marked diffuse changes, or there is exudative pleurisy upon the other side, or the pneumothorax is bilateral, the prognosis is rendered more gloomy, as a matter of course.

Under certain circumstances, the development of pneumothorax may improve the condition of the patient. Hérard, Czernieki, and others state that in some cases of phthisis the occurrence of pneumothorax improves the phthisical process and, in particular, diminishes the cough and secretion. In empyema it is not infrequently found that the perforation of the pus affords great temporary relief.

VI. TREATMENT.—In pure pneumothorax, which occurs unexpectedly when the lung is intact, the symptoms of collapse and asphyxia may demand active stimulation, viz., the administration of alcoholics, ethereal preparations, camphor, valerian, castoreum, etc. Cutaneous

irritants (mustard poultices and baths, alcoholic inunctions, dry-cups to the chest, etc.) usually afford the patient some relief.

In some of the cases, the dyspnoea is the result of nervous influences, caused by the suddenness of the accident. In such cases, the therapeutic measures mentioned will suffice. The dyspnoea generally grows gradually smaller, and there is hope that, after some time (perhaps several weeks), the air in the pleural cavity may be entirely absorbed. As a rule, the patient will experience great relief from the administration of narcotics (morphin. hydrochlor., 1.0; glycerin. pur., aq. destil., aa 15.0. M. D. S.  $\frac{1}{4}$ – $\frac{1}{2}$  syringeful subcutaneously).

If there are marked symptoms of displacement of adjacent organs, the dyspnoea must be attributed to this feature and to the excessive compression of the lung, and we must therefore endeavor to remove the air from the thorax. In open pneumothorax, operative interference would be useless, because air would again enter through the open fistula. The chances are better in closed than in valvular pneumothorax, because, in the latter, the air of the pleural cavity, after it has been rarefied as the result of the operation, will re-enter the thorax, and make another operation necessary.

As a rule, it will be sufficient to merely puncture the thorax and allow the gas to escape spontaneously. The trocar should be armed with a rubber tube, the end of which is placed under water, in order to prevent the aspiration of air into the chest during unexpected, vigorous respiratory movements. If there is an open valvular fistula, the operation must be repeated as soon as sufficient air has re-entered the pleural cavity to give rise to dangerous asphyxia from displacement of the organs. In closed pneumothorax, we may try, if necessary, to aspirate enough air to make the pressure within the pleural cavity lower than the atmospheric pressure.

If a pneumothorax is complicated with a fluid pleurisy, we should make an exploratory puncture (if the lungs are intact), in order to determine the character of the fluid. This will often be found purulent, and the indications are then the same as in empyema. The same indications hold good if an empyema has given rise to pyopneumothorax. If the exudation is serous, thoracentesis should be employed as in serous pleurisy. In the latter event, however, puncture is necessary only when life is in danger, while in pyopneumothorax it should be performed as early as possible.

When hydropneumothorax complicates pulmonary phthisis, operative interference should be delayed until imminent danger is impending. The results of the operation are very unfavorable on account of the primary disease. It results not infrequently in acute exacerbations of the phthisical process.

### 3. *Hydrothorax.*

I. ETIOLOGY. —Hydrothorax is an accumulation of oedematous fluid in the pleural cavity, *i. e.*, the fluid is a transudation. It is almost always associated with oedema of other localities, particularly the extremities.

The oedema is generally the result of stasis, in consequence of diseases of the heart or lungs.

Oedema of the pleural cavity develops as soon as the flow of blood from the superior vena cava to the heart is obstructed, and the blood in



the venæ azygos and hemiazygos presents an abnormally high pressure. Local affections of these vascular tracts (compression and thrombosis by mediastinal tumors) give rise, in rare cases, to stasis œdema of the pleural cavity.

In a second group of cases, a diminished amount of albumin in the blood gives rise to hydrothorax (Bright's disease, cancer, malarial and syphilitic marasmus, chronic diarrhœa, dysentery, leukæmia, cachectic conditions in general). The experiments of Cohnheim and Lichtheim render it probable that, in such cases, the diminution in the amount of albumin produces a nutritive disturbance of the walls of the vessels which makes them abnormally permeable.

Hydrothorax following exposure or certain exanthemata (particularly scarlatina), without the existence of nephritis and albuminuria, must be attributed to changes in the walls of the blood-vessels.

Occlusion of the lymphatics very rarely gives rise to the disease because the collateral branches of the lymphatic system are very well developed. Fraentzel, however, observed this affection as the result of compression of the thoracic duct.

It develops very frequently during the death struggle. This agonal hydrothorax is the result of circulatory disturbances, following irregular and feeble action of the heart.

From the remarks just made, it is evident that hydrothorax is merely a symptom, not an independent disease.

II. ANATOMICAL CHANGES.—A clear serous fluid is usually found in both pleural cavities. The fluid is amber-yellow or greenish-yellow in color, and is fluorescent; after a prolonged death struggle it is sometimes tinged with blood. It is usually thin and watery, and not infrequently contains delicate, light-gray clots. Crystalline shining deposits of cholestearin are sometimes found in old transudations. A cloudy and very flocculent fluid always indicates an inflammatory origin. Its reaction is alkaline, and its specific gravity varies from 1.009 to 1.012. In cases of heart disease the specific gravity may reach 1.020 to 1.023.

As a rule, a pleural fluid is a transudation if the specific gravity is less than 1.015, an exudation if it exceeds 1.018.

If transudations are present in various organs, chemical examination shows that the pleural fluid contains the largest proportion of solid constituents; then follow the peritoneum, cerebral ventricles, and subcutaneous cellular tissue. The proportion of solid matters varies considerably in individual cases of hydrothorax, as is shown by the following analyses :

	Schmidt.	Hoppe-Seyler.	Scherer.
Water, . . . . .	963.95	957.59	935.52
Solid matters, . . . . .	36.05	42.41	64.48
Organic matters, . . . . .	28.50	..	..
Fibrin, . . . . .	..	..	0.62
Albumin, . . . . .	..	27.82	49.77
Ethereal extract, . . . . .	..	14.59	2.14
Alcoholic extract . . . . .	..		1.84
Watery extract, . . . . .	..		1.62
Inorganic salts, . . . . .	7.55		7.98

Ewald found carbonic acid, oxygen, and nitrogen present in hydrothorax in the following proportions:

$$\begin{aligned}\text{CO}_2 &= 74.0 - 84.0 \\ \text{O} &= 0.29 - 1.01 \\ \text{N} &= 1.01 - 2.47\end{aligned}$$

Microscopical examination shows a few desquamated, swollen, partly fatty endothelium cells, a few lymphoid cells, red blood-globules, and granular cells.

In some cases only a few tablespoonfuls of fluid are present, in others as much as fifteen to eighteen pounds has been observed. The amount of fluid in the right pleural cavity is often greater than that in the left.

As a rule, the fluid is freely movable; its mobility is impaired only when old pleuritic adhesions are present.

The pleura is not infrequently of an opaque white color. The subserous connective tissue is often swollen as the result of œdematous imbibition.

The lungs are more or less compressed. In marked cases they may be converted into a firm, non-aërated mass of a reddish or grayish-brown color, situated near the spinal column. In almost all cases they may be distended with air, and permanent obliteration of the alveoli is observed only after the hydrothorax has lasted a long time.

The heart, liver, spleen, and diaphragm may be displaced.

III. SYMPTOMS AND DIAGNOSIS.—The subjective symptoms are almost always the result of compression of the lungs and displacement of the heart, and consist in the main of increasing dyspnœa, cyanosis, acceleration and feebleness of the pulse.

The objective symptoms alone justify a positive diagnosis. In many respects the local changes are similar to those of fluid pleurisy.

On inspection we not infrequently notice dilatation of the thorax, though not so marked as in fluid pleurisy, because the pressure of the fluid is less and the thoracic walls and diaphragm offer greater resistance. The intercostal spaces are often broader than normal, but rarely effaced. The respiratory excursions of the thorax are diminished. If the hydrothorax exerts equal pressure on both sides of the heart, this organ may not be displaced. At all events the displacement is not so marked as in pleurisy. There is not infrequently a considerable displacement of the liver and spleen.

In hydrothorax, as in pleurisy, the vocal fremitus is diminished or abolished over the fluid, and the feeling of resistance over the thorax is increased.

Dulness is present in both diseases, but in hydrothorax it usually changes with the movements of respiration and also upon changing the position of the body, the level of the fluid always remaining horizontal. The dulness is usually bilateral, though it is often unilateral at the onset, appearing first upon that side on which the patient is accustomed to lie. The disease frequently begins upon the right side, because most patients assume right lateral decubitus. This is the result of purely local disturbances in circulation. Indeed, a change in position not infrequently causes the disappearance of the hydrothorax upon one side and its appearance upon the other side of the chest. Constant unilateral hydrothorax is usually observed only when one pleural cavity is obliterated. This is often noticed in pulmonary phthisis.

On auscultation, vesicular breathing is found diminished or abolished, but bronchial breathing is not observed so frequently as in pleurisy. It is most apt to be heard in the interseapular space, in which the compressed lung is situated. Egophony is heard at the upper level of the fluid. I have also observed Baccelli's phenomenon in a number of cases. Bronchophony is diminished.

When the differential diagnosis between pleurisy and hydrothorax is doubtful, we should take the etiology into consideration and also make

an exploratory puncture. The specific gravity of the fluid in hydrothorax is almost always below 1.015. Hydrothorax is not infrequently complicated, after a while, with pleurisy. Pleuritic pains are then felt, the fluid removed on puncture becomes cloudy and flocculent, and its specific gravity increases. The disease often lasts many weeks or months, and presents exacerbations and remissions.

IV. PROGNOSIS AND TREATMENT.—The prognosis depends mainly on the primary affection. Even if the latter is incurable, but is capable of improvement, the fluid may be absorbed, although it generally returns and finally causes or hastens the fatal termination.

The treatment is the same as that of the primary disease (diuretics, drastics, diaphoretics, tonics). Puncture and aspiration should be performed only when the amount of fluid is a direct source of danger.

Care should be taken to prevent the entrance of air during the performance of aspiration. The operation must often be repeated. In one case, Ziemssen repeated it sixteen times in three and a half months.

#### 4. *Hemothorax.*

1. Hemothorax is an accumulation of blood in the pleural cavity. It may be the result of traumatism or of the following diseases: rupture of an aortic aneurism into the pleural cavity (usually the left), destruction of the wall of the aorta by ulceration, ulceration of the pulmonary veins and venæ cavae, rupture of varicose veins in the pleura, caries of the ribs which has caused perforation of the intercostal arteries, destruction of lung tissue in phthisical and gangrenous processes and in extensive pulmonary apoplexy.

2. The pleural cavity may contain blood alone or blood and air (hæmopneumothorax). The blood, which may remain fluid for a long time owing to the influence of the pleural endothelium, is sometimes absorbed very rapidly, occasionally within a few days. In some cases the hemothorax is followed by a secondary pleurisy.

The symptoms are those of fluid pleurisy. In making the differential diagnosis, we must take into consideration the etiology and those signs which are indicative of internal hemorrhage, viz., pallor of the skin, depression of the bodily temperature, syncopal attacks, a small, frequent pulse, etc.

3. The prognosis depends in two ways upon the amount of blood exuded: in the first place, because a great loss of blood may be the direct cause of death, and in the second place, because it compresses the lung and displaces the heart, thus giving rise to the danger of suffocation.

4. The indications for treatment are: to maintain the strength by the use of stimulants, to check further hemorrhage by the application of an ice-bag and subcutaneous injections of ergotin, to remove the blood by puncture if its excessive quantity proves dangerous to life.

#### 5. *Chylothorax.*

Quincke and v. Thaden have described cases of chylothorax, *i. e.*, an accumulation of chyle in the pleural cavity. In both cases, the disease was the result of injury, the chyle probably escaping from the torn thoracic duct; the chyle was mixed with blood. The fluid had a fatty, milky appearance, and, after standing, a thick layer of cream formed upon the surface. It had an alkaline reaction, contained sugar and a



sugar-forming ferment. In v. Thaden's case, the fluid contained 0.43 per cent of sugar and 3.71 per cent of fat. It also contained a few lymphoid corpuscles. Both cases affected the right side and were treated unsuccessfully by puncture.

At the autopsy, white creamy coagula were found upon the layers of the pleura.

Chylothorax should not be mistaken for other exudations which contain fat. This is observed particularly in cancer of the pleura, but also independently of it.

The clinical history, course, and treatment are similar to those of hemothorax.

## 6. *Tumors of the Pleura.*

1. **TUBERCLES.**—A general development of tubercles upon the pleura occurs in general miliary tuberculosis. In some cases we find local tuberculosis of the pleura, especially over the cheesy, tuberculous foci in the lungs and in cheesy, tuberculous degeneration of the bronchial glands. Finally, primary tuberculosis of the pleura is sometimes secondary to pleurisy. In the latter event, gray transparent nodules develop mainly in the false membranes, in which they may be densely aggregated.

Gray miliary tubercles are generally found, more rarely cheesy nodules. The latter may be collected into large masses and nodules. In general tuberculosis both pleuræ are strewn with miliary tubercles; when the process develops after pleurisy, it is sometimes found only on that side of the chest which had been previously affected.

In the majority of cases, miliary tuberculosis of the pleura remains latent during life. Juergensen states that he has heard in several cases a peculiar soft rubbing, which is more gentle and soft than a pleuritic friction murmur. If tubercular pleurisy develops, the fluid exudation is often hemorrhagic, and the symptoms are those of fluid pleurisy. Treatment is useless.

2. **CANCER.**—Cancer of the pleura is almost always secondary. The primary cancer is situated most frequently in the lungs, mammary, bronchial and axillary glands, on the peritoneum, or other abdominal organs. Sometimes the cancer spreads by direct proliferation from the vicinity, sometimes it is far removed from the primary cancer and forms distinct metastases.

Medullary cancer is most frequent, but scirrhus, colloid, and even epithelial cancer have been observed. Pleural cancer is more often unilateral than bilateral. It appears sometimes as almost miliary nodules which remind us of tuberculosis, in other cases large masses of cancer may attain the size of a man's head, and give rise to compression of the lungs and adjacent organs.

Occasionally we find a few scattered nodules upon the pleura with a peculiarly flattened surface, in other cases there is a diffuse cancerous infiltration, which may give rise to considerable thickening of the pleura. Small, miliary nodules of cancer are sometimes seen along the course of the pleural lymphatics.

The pleural cavity very often contains fluid, generally transudation, more rarely exudation (carcinomatous pleurisy). The latter may be purulent, ichorous or bloody, and appears especially when ulcerative processes have formed in the cancerous proliferations.

The diagnosis of pleural cancer is very difficult and in almost all cases a probable diagnosis can alone be made.

It is said that small nodules may give rise to circumscribed friction murmurs, and this should arouse our suspicion if cancer is demonstrable in other organs.

Some patients suffer from an extremely annoying cough which is a constant source of distress. Examination of the chest often gives entirely negative results, and the presence of cancer in other organs alone arouses suspicion.

If the growth is complicated with hydrothorax or pleurisy, the signs of fluid in the pleural cavity will be manifested.

Extensive cancer of the pleura will itself give rise to dulness on percussion. Vocal fremitus may be diminished or abolished, though this is not always the case. If the tumor is very large, it causes dilatation of the thorax and displacement of adjacent organs. The lungs may also be compressed and pushed against the spinal column, so that the respiratory murmur is diminished or becomes bronchial in character. The growth sometimes perforates the muscles and appears under the skin. Pressure upon the aorta or venæ cavæ may give rise to various disturbances of circulation, and disorders of deglutition may appear if the œsophagus is compressed. The patients often complain of a feeling of weight and pressure within the chest, which may be intensified into severe pain. Cough and dyspnoea are usually observed.

The disease may be mistaken for :

*a.* Fluid pleurisy or hydrothorax. This is particularly true if the thorax is dilated and the adjacent organs are displaced. It should be noted that, in the case of tumors, the dulness is often extremely irregular and that its boundaries do not change on changing the position of the body.

*b.* A suspicion of aortic aneurism may be aroused if the cancerous mass is situated between the aorta and anterior chest-wall, and pulsating movements are thus conveyed to the tumor. In the latter, however, the movement is always that of elevation and depression, in aneurism the pulsation takes place on all sides. If murmurs are present in pleural cancer, they are simply systolic stenosis murmurs over the compressed aorta, but the systolic and diastolic murmurs of aneurism are absent. The etiology should also be taken into consideration.

*c.* The disease may also be mistaken for cheesy changes in the lungs. In the latter affection, the vocal fremitus is increased, and very loud bronchial breathing is usually heard. Dilatation of the thorax and signs of displacement are always absent.

*d.* It can rarely be determined whether a tumor, which has been diagnosed, grows from the pleura alone or from adjacent organs (lungs, mediastinum, etc.).

The prognosis is absolutely unfavorable. The treatment must be confined to the amelioration of the sufferings of the patient.

3. Sarcoma of the pleura is rarer than carcinoma. The remarks made concerning cancer also hold good concerning sarcoma.

4. Enchondroma and dermoid cysts of the pleura are anatomical rarities which do not possess any clinical significance.

### *7. Animal Parasites of the Pleura.*

1. After ulceration has taken place, echinococci may enter the pleural

cavity from the liver or lungs, or, in rare instances, may develop primarily in the pleural cavity. They may attain the size of a man's head and give rise to a corresponding dilatation (usually circumscribed) of the thorax and displacement of the liver and heart. They may also compress the lungs to such an extent as to cause danger of death from suffocation. Purulent pleurisy not infrequently develops secondarily.

Pain, cough, and dyspnoea are noteworthy symptoms. If the vesicles are sufficiently large, we will find dilatation of the thorax, displacement of adjacent organs, feeble or abolished vocal fremitus, dulness and abolition of the respiratory murmur. The disease is readily mistaken for fluid pleurisy; special attention should be paid to the irregular course of the boundaries of dulness. The differentiation from solid pleural tumors may be very difficult. This is often decided by an exploratory puncture, which gives vent to a clear fluid of low specific gravity, destitute of albumin, and in which hooks, scolices, and shreds of the vesicle are occasionally found (vide page 333, Fig. 88). If secondary empyema develops, its true course often remains concealed. The vesicles sometimes project so forcibly through the chest-walls, that operative interference becomes necessary.

The treatment is purely surgical: incision and removal of the cysts.

2. Cysticerci have been found in the pleural cavity, but the vesicles are usually so small that the diagnosis is impossible.

## PART VII.

### DISEASES OF THE PULMONARY ARTERY.

#### 1. *Aneurism of the Pulmonary Artery.*

1. This is an exceedingly rare affection. Crisp found only four cases among nine hundred and fifteen cases of aneurism. It is generally situated upon the main trunk, and may be either sacculated or spindle-shaped. As a rule, it is associated with arterio-sclerotic changes in the wall of the vessel, and in Wolfram's case these could be followed into the finest ramifications. Endocarditic changes are often observed, sometimes upon all the valves. Buchwald and Foulis found it associated with patency of the ductus Botalli. Nothing is known concerning its essential causes. It sometimes appears at an early age (seventeen years in one case, nineteen years in another).

2. The symptoms are not characteristic, and the disease is often unrecognized. If the aneurism is situated upon the origin of the artery, we must look for hypertrophy and dilatation of the right heart, dulness and prominence (which pulsates in all directions) in the second left intercostal space, a systolic and also a diastolic murmur, most distinct over the region of dulness. Dyspnoea, pallor, cyanosis, and hæmoptysis have also been observed.

As a rule, however, the phenomena are not so simple. Both ventricles are often dilated and hypertrophied; murmurs are heard not infrequently over other valves, so that they are localized with difficulty. Pulmonary aneurisms may often be mistaken for aortic aneurisms, and in such cases the changes in the pulse merit special attention. Death is generally the result of suffocation, secondary pericarditis, or hemorrhage from rupture of the aneurism.



3. The prognosis and treatment are the same as in cases of aortic aneurism.

## 2. *Embolism of the Pulmonary Artery.*

I. ETIOLOGY.—Embolism of the pulmonary artery occurs not infrequently. In the large majority of cases the embolus consists of fibrinous deposits from the blood, more rarely of particles of tumor, echinococcus vesicles, fat, or bubbles of air.

Fibrinous emboli are usually derived from thrombi of the right heart or peripheral veins. They have also been observed after right endocarditis and valvular lesions, cardiac thrombosis, marantic venous thrombosis, in child-bed when thrombi form in the peri-uterine or peri-vaginal veins, in uterine and ovarian tumors when thrombi form in their veins, after operation upon the genital organs, in inflammation of the prostate, intestinal ulcers, etc. Fractures of bone, cutaneous phlegmons, furuncles, varices, bed-sores, contusions of the skin may also give rise to embolism of the pulmonary artery if thrombosis has developed near the site of injury. The disease may also be secondary to thrombosis of the cerebral sinuses, lesions of the bones around the auditory canal, or aneurism of the pulmonary artery. The dislodgment of the thrombus sometimes occurs spontaneously, sometimes it is the result of sudden sitting up, careless pressure upon a peripheral venous thrombus (for example, during examination by the physician, etc.).

In occlusion of the pulmonary artery by particles of neoplasm or echinococci, the embolic material may be derived from the right heart, as well as the periphery of the body.

Fatty embolism possesses greater interest for the surgeon than the physician because it is usually the result of fracture, the medulla of the bones being forced into the surrounding tissues and then absorbed by the veins. According to Hamilton, Sanders, and Starr, a deposit of fat may take place in the blood (lipemia) in diabetes mellitus, and this may occlude the pulmonary vessels and produce diabetic coma. The occurrence of fatty emboli is also mentioned in Bright's disease, grave jaundice, rupture of a fatty liver, purulent degeneration of cardiac thrombi, large abscesses, and puerperal inflammations of the uterus.

The conditions for the production of air emboli are furnished when air is sucked into the vessels after injury to large veins (particularly in the neck) or when air-containing organs are brought into connection with the lumen of the veins as the result of ulcerative processes. The injection of fluid containing air into the female genitals may also be followed by embolism.

II. SYMPTOMS.—The development of pulmonary embolism is not infrequently the cause of sudden death. The patients cry out aloud and then fall lifeless to the ground. Many sudden deaths during the puerperal state are the result of this affection. It is especially apt to occur if the trunk or one of the two main branches or even one of the larger ramifications is suddenly occluded completely.

If the obstruction is not complete, or if smaller branches are affected, the disease may be protracted over several hours or even days. The patients suddenly experience indescribable anguish and dyspnoea; the respirations are accelerated, gasping, labored; the face is distorted; the integument is pale, cyanotic, cool, and covered with clammy sweat; the pulse is imperceptible, the heart's action is accelerated and irregular; the stenosis of the pulmonary artery by the embolus is often associated

with a murmur of stenosis; consciousness is gradually clouded and the vital energies fail; death is not infrequently preceded by convulsions. The symptomatology is explained by the fact that the embolism is followed by thrombosis which gradually extends and becomes more complete. Hence disturbance of the pulmonary interchange of gases and anæmia of the brain.

Finally, the phenomena of hemorrhagic infarction must be mentioned among the results of pulmonary embolism (vide page 114).

Certain emboli also possess infectious properties. These depend chiefly on the character of the thrombus from which the embolus is derived. Hence, embolism may result in pneumonic, purulent, ichorous changes in the lungs.

III. ANATOMICAL CHANGES.—If the emboli are formed of firm masses, they are found usually at the bifurcation of the vessels. Long fibrinous clots sometimes extend into several adjacent vessels. If the patient remains alive, a wedge-shaped infarction is usually produced (vide page 252).

Small infarctions may be entirely absorbed. In other cases, they retract and are converted into a cicatrix, in still others, they become dry, cheesy, and even calcareous.

If the emboli are derived from tumor particles or echinococcus vesicles, they may continue to proliferate in the lungs.

In fatty embolism, it is particularly the pulmonary capillaries which are filled with drops of fat. These may also pass into the interstitial connective tissue of the lungs or into the pulmonary veins, and may then give rise to embolism of the cerebral capillaries or other capillaries of the periphery of the body. The urine not infrequently contains fat—an evidence that fatty embolism has occurred in the vessels of the kidneys. Fatty embolism is sometimes followed by hemorrhagic infarction.

Air emboli of slight extent are soon absorbed by the blood; if more extensive, the finer vessels are found filled with air bubbles.

IV. DIAGNOSIS.—The recognition of pulmonary embolism is often impossible. In cases of heart disease, embolism of the pulmonary artery should be suspected if hæmoptysis occurs, though this may be the result—but much less frequently—of simple rupture of the vessels, in consequence of stasis of blood. This also holds good concerning the recognition of pulmonary embolism during the course of peripheral venous thrombosis. In other words, a great deal depends upon the proof that embolic material is situated somewhere at the periphery of the body. This is also true concerning fatty and air emboli. In the latter affection, gurgling sounds have been heard occasionally in the heart, so long as air was present in the right half of the organ. The embolism sometimes occurs in a latent manner, and is manifested later as pneumonia, abscess, and pulmonary gangrene.

V. PROGNOSIS.—The prognosis is always grave, especially when the emboli possess infectious properties.

VI. TREATMENT.—It is often necessary to make vigorous use of stimulants. Venesection has been attended with very little effect. In hæmoptysis the treatment described on page 256 is indicated.

### 3. *Thrombosis of the Pulmonary Artery.*

Thrombosis of the pulmonary artery develops under the same conditions as in other vessels. Long-standing diseases, conditions of exhaus-

tion, the protracted maintenance of the same position of the body, are the most frequent causes. In all probability, thrombosis of the pulmonary artery is more frequent than is generally believed, and not a few cases of supposed embolism are really instances of thrombosis. As a matter of course, a thrombus will be followed by the same effects as an embolus, *i. e.*, by an hemorrhagic infarction. The slower the development of the obstruction, the greater is the chance that the circulatory disturbances will be compensated by the adjacent capillary tracts, and hence an hemorrhagic infarction is less apt to develop in thrombosis than in embolism. In other respects, the remarks made in the previous section hold good concerning thrombosis.

#### 4. *Stenosis of the Pulmonary Artery.*

1. Stenosis of the trunk, or of one of the main branches of the pulmonary artery may be the result of various causes, such as compression of the vessels by an aortic aneurism, mediastinal tumor or abscess, swollen lymphatic glands, or retraction-processes in the mediastinum (cicatricial mediastino-pericarditis) or lungs. It has also been observed as the result of retraction of bronchial glands. In rarer cases, it is owing to thickening of the tunica intima of the vessels. In individuals who possess a yielding thorax we are not infrequently able, by pressure with the stethoscope, to produce a stenosis-murmur in the pulmonary artery, especially when the vessel is in direct contact with the thorax to an unusual extent on account of retraction of the upper lobe of the left lung.

2. The symptoms consist chiefly of a systolic murmur over the pulmonary artery, and intensification of the diastolic pulmonary sound. A diastolic murmur is sometimes heard, on account of excessive dilatation of the pulmonary orifice, and relative pulmonary insufficiency. In addition, we find dilatation and hypertrophy of the right ventricle, palpitation, dyspnoea, and cyanosis. The systolic murmur is sometimes heard in the interseapular space. The treatment is the same as that of stenosis of the pulmonary orifice (*vide* page 134).

### PART VIII.

#### DISEASES IN THE MEDIASTINUM.

##### 1. *Mediastinal Tumors.*

I. ETIOLOGY AND ANATOMICAL CHANGES.—Among the tumors of the mediastinal space, cancer and sarcoma are first in point of frequency. Lipoma, fibroma, dermoid cysts, osteoma, cysts, teratoma myomatodes (a cyst-like tumor composed of striated muscular fibres), echinococcus, have also been found in this region.

The cancers are usually extremely soft and rich in cancer juice (medullary cancer); firm scirrhus is rarer. Horstmann described a case of epithelial cancer.

The sarcomas are often of the lymphoid variety, the starting-point being the lymphatic glands of the mediastinal cellular tissue.

Lipomata and fibromata are the result of excessive hyperplasia of the mediastinal adipose and connective tissue; osteomata, in the shape of



outgrowths from the inner surface of the sternum, have been described as sequelæ of scrofula and syphilis.

The dermoid cysts are the results of aberration of fœtal germinal tissue. They may contain hair, teeth, and pieces of bone.

The mediastinal lymphatic glands and cellular tissue form the starting-point of the tumor in many cases. In other cases they start from organs which are situated in the mediastinal space or are adjacent to it. For example, mediastinal tumors may be the result of degeneration of the thymus gland (carcinoma, sarcoma, fibroma, even dermoid cysts). Or tumors which first developed upon the pericardium involve the mediastinal tissues to such an extent that the symptoms of mediastinal tumors occupy the foreground.

These tumors are much more frequent in men than in women. Among thirty-five cases collected by me, twenty-six occurred in males, nine in females.

Age also exerts an influence upon their development; the majority of cases are found from the twentieth to the thirtieth years.

	Cases.		
1-10 years.	4	=	11.8 per cent.
10-20 "	5	=	14.7 "
20-30 "	9	=	26.5 "
30-40 "	7	=	20.6 "
40-50 "	5	=	14.7 "
50-60 "	3	=	8.8 "
60-70 "	1	=	2.9 "
	—		
	34		

Many authors maintain that syphilis has no effect on the development of mediastinal tumors, but according to French writers the osteomata, which grow from the inner surface of the sternum, must be attributed to syphilis. Scrofula also exerts an influence in certain cases, particularly in childhood and in lympho-sarcoma of the mediastinum. The lymphatic glands of the mediastinum, as of other localities, may also undergo hyperplasia in leukæmia. This is also true of pseudoleukæmia (Hodgkin's disease).

The patients sometimes state that the first symptoms developed after great exertion or external injury, and the disease is not infrequently known to run a very rapid course under such circumstances.

Tumors are very often found in other internal organs in addition to the mediastinum. If the organs are remote, metastasis has evidently occurred.

II. SYMPTOMS.—The most prominent symptoms of mediastinal tumors are those of pressure upon adjacent organs. These may be the result of very small tumors, and may then constitute the sole phenomena.

Large mediastinal tumors are manifested by prominence, abnormal dulness, and displacement of adjacent organs.

In tumors of the anterior mediastinum the prominence is observed most frequently over the manubrium sterni and upper half of the body of the sternum, but not infrequently the adjacent intercostal spaces take part in the swelling. The integument of this region is often glossy and destitute of folds. If the prominence is slight, it is not infrequently better recognized by oblique illumination. Erosion of the sternum and ribs may occur, or the bony parts may undergo the neoplastic degeneration. Under such circumstances, the prominence often has an elastic, yielding, occasionally fluctuating feel; it may also undergo pulsations

which have been conveyed to it by the underlying arteries. If the tumors extend into one or the other half of the thorax, the entire chest on that side may be abnormally dilated.

There is dulness on percussion over that portion of the chest-wall with which the tumor is in immediate contact. Tumors of the anterior mediastinum usually give rise to abnormal dulness over the upper part of the sternum; tumors of the posterior mediastinum produce dulness upon the posterior wall of the thorax. In the former event the dulness is not infrequently continuous with cardiac dulness, and may be regarded as evidence of aneurism or pericarditis.

The heart is very often displaced. In tumors of the anterior mediastinum the displacement is generally downwards and to the left, so that the apex beat is below the fifth intercostal space, and is often outside of the mammary line. Tumors of the posterior mediastinum usually push the heart against the anterior chest-wall, so that the cardiac movements are often visible in several intercostal spaces. If the tumor fills the entire mediastinal space, the liver, particularly the left lobe, may also be displaced. The lower border of the liver, in the median line, is then found below a point midway between the ensiform process and umbilicus. Displacement of the spleen is occasionally observed.

Evidences of pressure are manifested by the vessels, respiratory organs, pneumogastric, sympathetic, and the œsophagus.

Compression or occlusion of one or both innominate veins is observed most frequently. The occlusion may be the result of perforation of the walls of the vein by the tumor, and its proliferation within the lumen of the vein. If the occlusion or stenosis is unilateral, there is unilateral œdema of the face, neck, and arm. The subcutaneous veins of the thorax over the situation of the deeper veins are enlarged and tortuous. If both innominate veins are affected, these phenomena are observed upon both sides of the body. The compression and occlusion are sometimes confined to a subclavian vein. We then find œdema, livid discoloration, coldness of the corresponding arm, and dilatation and tortuosity of its subcutaneous veins. These phenomena may also be produced by the pressure of carcinomatous or sarcomatous axillary lymphatic glands upon the subclavian vein. Pressure upon the azygos and hemiazygos veins will be manifested by marked dilatation and tortuosity of the subcutaneous veins of the abdominal walls, and by œdema in the same locality.

The large arterial trunks offer much greater resistance to the pressure, but abnormal stenosis-murmurs are sometimes produced in them. According to the situation of the tumor, they will be heard at the origin of the aorta and pulmonary artery, or upon the posterior surface of the thorax, along the spinal column. Compression of individual arterial trunks may give rise to abnormal retardation and feebleness of the radial pulse.

The respiratory apparatus presents manifold disturbances. The lungs are not infrequently compressed to such an extent that the danger of suffocation arises. This is especially true if the parenchyma of the lung is infiltrated with the tumor. In other cases a bronchus or the trachea is compressed, and the tumor may proliferate into these parts. Such conditions are recognized by marked dyspnea, inspiratory retraction of the intercostal spaces, enfeebled thoracic respiratory movements, diminished vocal fremitus, deep tympanitic percussion note, rough and feeble respiratory murmur. According as these symptoms are unilateral or bi-

lateral, we must assume compression of a bronchus or of the trachea, more rarely of both bronchi.

The feeling of dyspnœa is usually associated with a definite passive position of the body, generally a sitting or elevated lateral decubitus, more rarely abdominal decubitus.

Paroxysms of suffocation often occur, and are attributed to temporary irritation of the pneumogastrics. These attacks resemble asthma, and are characterized by dyspnœa, the objective signs of dyspnœa and stridorous breathing. The duration and frequency of the attacks vary greatly. They are sometimes provoked by certain positions of the body.

Disturbed innervation of the vocal cords may often be recognized with the aid of the laryngoscope. There is usually paralysis of one or both recurrent laryngeal nerves (vide page 196). In one case Riegel observed stenosis of the trachea. If both vocal cords are paralyzed, the voice becomes aphonic; if one cord is paralyzed, the voice becomes higher in pitch, and sometimes falsetto.

In a case reported by Anderson, changes in the voice occurred only during certain positions of the body. The voice became hoarse as soon as the patient turned the head to the left or lay on the back with the head bent strongly backwards. Anderson assumes that the pressure on the recurrent laryngeal nerve varied in different positions of the body.

In some autopsies the tumor has been found to proliferate into the neurilemma of the pneumogastric.

Paralysis of the recurrent laryngeal nerve is also dangerous, because it interferes with deglutition. The paralysis of the epiglottis and the imperfect closure of the rima glottidis allow the entrance of food into the larynx and lungs, where it gives rise to inflammatory or gangrenous processes.

Irritation or paralysis of the pneumogastric is sometimes shown by the effects on the movements of the heart. The contractions of the heart are irregular and very slow (irritation), or they are extremely accelerated (paralysis). If the fibres supplying the muscular coat of the œsophagus and stomach are affected, attacks of œsophageal spasm, singultus, and vomiting are produced.

Irritation or paralysis of the cervical sympathetic is shown by changes in the pupils. They are often unequal, one being unusually narrow, the other extremely wide.

Rosbach carefully studied the pupillary changes in three cases. In the first the pressure of the tumor, which projected above the clavicle, produced dilatation of the pupil. The dilatation increased with the pressure, and, at the same time, the pulse became irregular, smaller, and slower. The same phenomena were observed in the second case, except that the pulse became more rapid. On deep inspiration the pupils became dilated, and remained so until after the beginning of expiration; they reacted to light. In Horstman's case, contraction of the left pupil was produced by a venous thrombus which pressed upon the left third nerve.

Exophthalmus and goitre have been observed in a few cases.

Compression of the œsophagus produces permanent disturbances of deglutition, and, if the tumor is small, this may constitute the sole symptom. Large tumors may give rise to complete occlusion of the œsophagus, and to death from inanition.

The subjective symptoms are usually of no trifling character. The



patients complain not infrequently of a burning, sticking pain beneath the sternum. This is either constant or paroxysmal (especially after exertion). Neuralgic pains in the limbs have also been observed as the result of pressure on the nerve trunks by large, metastatic tumors. Pains in the spinal column are sometimes noticed, particularly if the vertebrae have been implicated in the new growth. Compression of the spinal cord may result in paralysis.

The patients complain not infrequently of vertigo, ringing in the ears, *muscae volitantes* and similar symptoms, which must be attributed to cerebral congestion. Insomnia develops in some cases.

In many cases the patients very rapidly assume a cachectic appearance, and the blood is found not infrequently in a condition of leukocytosis. The cough is often very distressing, the sputum scanty, catarrhal, often tinged with blood. Death has even been known to result from hæmoptysis.

The expectoration of hairs has been observed in several cases of dermoid cysts in the mediastinum, which had ruptured into the bronchi.

The duration of the disease depends upon the rapidity of growth of the tumor and the degree of interference with adjacent organs. Cases have been known to last five to seven years, others run their course in a few weeks. According to Destord, the average duration is three to seven months.

Death is sometimes the result of general marasmus: the patients become dropsical, lose their strength, albuminuria sets in, and death follows from exhaustion. In other cases, the fatal event is the result of compression of adjacent organs. Thus, compression of the lungs, trachea, or main bronchi may produce death by suffocation. If the recurrent nerves are paralyzed, "foreign body" pneumonia, or gangrenous processes in the lungs terminate the scene. Occlusion of the œsophagus is sometimes the cause of death from inanition. In rarer cases it is the result of cerebral hemorrhage. In many cases the immediate cause of death is some accidental complication, such as pneumonia, pleurisy, or pericarditis.

III. DIAGNOSIS.—Small mediastinal tumors remain entirely latent if there are no symptoms of pressure on adjacent organs. In other cases, the latter alone lead to the diagnosis of the disease. The special symptoms are unilateral or bilateral paralysis of the recurrent laryngeals, difficulty in deglutition and inequality of the pupils. Under such circumstances, we must be on our guard against mistaking the disease for aortic aneurism; special attention should be paid to retardation of the pulse, vascular murmurs, and age. If there is difficulty in deglutition, the age of the patient must be taken into consideration, in order to exclude œsophageal cancer as an improbable cause. After exploring the œsophagus, the masses adherent to the sound should be carefully examined since they are apt to consist of small particles of the tumor, and are readily recognized under the microscope.

Tumors in the anterior mediastinum, which are large enough to produce abnormal dullness, may be mistaken for aneurism or pericarditis. Tumors of the posterior mediastinum must be differentiated from circumscribed pleurisy. If pleurisy is associated with the neoplasm, the difficulties are insurmountable. The disease is differentiated from pericarditis by the history, the relation of the apex beat to cardiac dullness, and the usually irregular shape of the area of dullness. The differentiation from aneurism may be difficult; attention should be paid to the

considerations previously mentioned. Enlargement of peripheral lymphatic glands or metastases in other internal organs favor the diagnosis of mediastinal tumor.

Great care must be exercised in the diagnosis of pulsating tumors. Unlike an aneurism, the tumor merely rises and falls; furthermore, the tumor gives a sudden impulse, while the aneurism gradually fills and pulsates.

Mediastinal tumors may usually be distinguished from mediastinal abscesses because the latter generally follow injury or exposure, and run an acute course, attended with febrile movement.

The anatomical character of the tumor cannot always be determined with certainty. Rapid growth and implication of the axillary, supraclavicular, or inguinal glands favor the diagnosis of cancer or sarcoma. Age must also be considered in making a diagnosis of carcinoma; sarcoma or lymphosarcoma is to be looked for if scrofula, leukæmia, or pseudoleukæmia is present.

IV. PROGNOSIS AND TREATMENT.—The prognosis is unfavorable, and the more rapid the growth of the tumor the earlier is the fatal termination.

The treatment is chiefly symptomatic. Very little can be hoped from the internal or external use of preparations of iodine. Arsenic may be administered in cases of sarcoma or lymphosarcoma. Koenig recently resected the sternum and successfully removed a mediastinal tumor.

## 2. *Inflammation in the Mediastinum. Mediastinitis.*

I. ETIOLOGY AND ANATOMICAL CHANGES.—Inflammation of the mediastinal connective tissue is not often observed. The inflammation may be acute or chronic, and situated in the anterior or posterior mediastinum. Acute mediastinitis has a tendency to pus formation (mediastinal abscess), while the chronic form results not infrequently in the formation of fibrous cicatrices which may interfere considerably with the organs situated in the mediastinum.

Mediastinitis is rarely primary; it has been observed after a blow on the chest and, it is said, after exposure. In the majority of cases the disease is secondary to inflammation of adjacent organs.

The mediastinum may become implicated in various processes of the sternum, ribs, or spine; pericarditis and pleurisy often extend directly to the mediastinal connective tissue. Mediastinitis may also result from the perforation of pus from the pericardial or pleural cavities, from a pulmonary abscess, from a cold abscess following spinal caries, suppurating lymphatic glands, or suppurative processes in the cellular tissue of the neck. Fraentzel reports a case in which the administration of chloride of iron produced a violent inflammation in the œsophagus and surrounding connective tissue; this extended downwards into the mediastinum where it produced fatal inflammation and suppuration.

Metastatic mediastinitis and abscess have been observed a number of times in infectious diseases and pyæmia.

In some cases, chronic mediastinitis seems to be the result of chronic pulmonary diseases (phthisis, emphysema, interstitial pneumonia, bronchiectasis).

In acute mediastinitis, the tissue is found reddened and swollen; either a diffuse purulent infiltration or an abscess is found to be present.

Chronic mediastinitis is characterized by thickening and the formation of cicatrices.

II. SYMPTOMS.—In acute mediastinitis, the general symptoms are fever, chill, and a feeling of malaise. The most prominent local symptoms are a distressing feeling of pressure and severe pain beneath the sternum. Gentle percussion over the sternum or pressure along the spine, according as the inflammation is situated anteriorly or posteriorly, also causes pain. The integument over the sternum is not infrequently hot, red, and oedematous. In addition, we notice palpitation and weakness of the heart, dyspnoea and terror, cough and expectoration of mucopus which is not infrequently tinged with blood, and disturbances of deglutition. If the inflammation resolves, these symptoms gradually subside.

If an abscess forms, areas of abnormal dulness appear upon the anterior or posterior surface of the chest, as in the case of tumors. Anteriorly they are found beneath the sternum, and project more or less to the side. If the abscesses attain considerable dimensions, symptoms of pressure upon adjacent organs make their appearance, especially dilatation and sinuosity of the cutaneous veins, oedema, and more marked disturbances of deglutition.

The pus often makes its way externally, perforating the sternum, and appearing as a fluctuating tumor beneath the integument. According to Dandé, the perforation takes place most frequently in the second intercostal space near the sternum. In a few cases, the pus has made its appearance in the loins or groins.

The pus occasionally ruptures into internal organs, with relative frequency into the pericardial or pleural cavities, a bronchus, lung, or the œsophagus. A very grave sequel is erosion of the large arteries (aorta, internal mammary, etc.), since this is inevitably followed by fatal hemorrhage.

Death often occurs with surprising rapidity after grave symptoms of collapse. Numerous dangers may arise, even when the disease is somewhat more protracted.

Chronic mediastinitis sometimes follows the acute form, sometimes develops as such from the start. We have previously referred to cicatricial mediastino-pericarditis (page 24). Fibrous cicatrices in the neighborhood of the œsophagus may give rise to the formation of traction-diverticula of the organ. These may cause disturbances of deglutition, and may also lead to perforation and death. The diverticula are the result of retraction of the cicatrices which pull upon the points of insertion into the walls of the œsophagus, and draw them outwards. Traction-diverticula of the air passages are rarely produced, because the cartilaginous walls offer sufficient resistance; broncho-stenosis is much more apt to be produced. Stenosis of the aorta, pulmonary veins and aorta, has also been observed. Finally, the recurrent laryngeal nerve may also be compressed and paralyzed.

III. DIAGNOSIS. PROGNOSIS. TREATMENT.—Acute mediastinitis is often concealed by the primary disease, and the chronic form can be recognized by its consequences only if other etiological factors can be excluded.

The prognosis of acute mediastinitis is very grave, and that of the chronic form is also serious.

Local treatment (ice-bag, leeches) must be adopted in the acute form. In addition, wine and nourishing diet are given to maintain the pa-



tient's strength. Trepanation of the sternum has been advised if the presence of pus in the mediastinum is suspected. When the pus points, an incision should be made under antiseptic precautions.

In chronic mediastinitis, the treatment must be purely symptomatic.

### 3. *Mediastinal Hemorrhages.*

Small extravasations into the mediastinal connective tissue are found in many conditions of blood dissolution (grave icterus, hemorrhagic exanthemata, infectious diseases, scurvy, etc.), but possess no clinical significance.

Extensive mediastinal hemorrhages may follow the rupture of large vessels, and, as a rule, a fatal termination rapidly ensues.

Finally, large hemorrhages may follow a blow, fall, or perforating wounds. The diagnosis then depends on the signs of internal hemorrhage (pallor, weak pulse, cool skin, faintness), on the acute development of abnormal dulness over the mediastinal space, and, at times, on the signs of compression of adjacent organs. Dandé states that pathognomonic ecchymoses appear in the lumbar region on the second or third day. The hemorrhage may prove fatal by its size or by the compression of adjacent organs, or it may lead to the development of a mediastinal abscess. The treatment consists of local antiphlogosis and the use of stimulants.

### 4. *Mediastinal Interstitial Emphysema.*

Some observations seem to indicate that the lungs may rupture near the hilus during bodily exertion, and that air may then enter the connective tissue between the pericardial pleura and the pericardium. In the cases described, a peculiar crackling sound was heard, which was associated with the cardiac movements, and was so loud that it could be heard throughout the whole apartment. In addition, pains were felt in the region of the apex beat. The symptoms disappeared in a few days.

## APPENDIX.

### *Diseases of the Thymus Gland.*

1. This gland is situated in the anterior mediastinum and covers the anterior surface of the pericardium and the origin of the large vessels. It extends upwards, occasionally to such an extent that it touches the thyroid gland, and downwards to the fifth costal cartilage. It increases in size until the second year of life, and then remains unchanged until the age of puberty. About the age of fifteen years it begins to undergo atrophy and fatty degeneration, and in adults it usually forms a fat-containing lump. In children, the gland may give rise to an area of dulness extending from the second to fourth costal cartilage along the left border of the sternum.

2. Hypertrophy of the thymus gland has been described a number of times, but it must be remembered that, under normal conditions, it presents great variations in size and weight. Kopp attributes spasm of the glottis to hypertrophy of this gland, but the two affections may exist independently of one another. The condition cannot be diagnosed during life.

The gland may persist to an advanced age.

3. Hemorrhages into the gland may be associated with hemorrhages into other organs. The extravasations vary from the size of a pin's head to that of a pea. They have been observed in asphyctic children and in conditions of blood dissolution.

4. Abscess of the gland, as a sequel of inflammation, was first observed by Dubois in congenital syphilis. The milky juice of the gland should not be mistaken for pus. The abscesses in this region may also develop independently of syphilis. Puerkhauer reports the case of a boy of four years who suddenly suffered from an attack of suffocation and died in a short time. The autopsy showed suppuration of the entire thymus gland, and the rupture of the pus into a bronchus.

5. Cheesy degeneration of the gland is said not to be infrequent, and to lead to meningeal tuberculosis. In some cases, the caseation is said to be connected with the process of involution of the gland; in others it is attributed to scrofula. Miliary tubercles have been found in the gland in cases of general miliary tuberculosis.

Cancer, sarcoma, and lymphosarcoma have been observed in the thymus gland in a number of cases. Dermoid and other cysts may also occur in this locality. If sufficiently large, the growth may give rise to the symptoms of a mediastinal tumor.

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